MEAN CORPUSCULAR VOLUME AS A MARKER OF ALCOHOL USE DISORDERS

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CERTIFICATE

This is to certify that the dissertation titled **"MEAN CORPUSCULAR VOLUME AS A MARKER OF ALCOHOL USE DISORDERS"** is the bonafide original work of **Dr.M.RAJA** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in APRIL 2014. The Period of study was from July 2013 to December 2013.

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DECLARATION

I, **Dr.M.RAJA** solemnly declare that dissertation titled **"Mean Corpuscular Volume as a marker of alcohol use disorders"** is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during July 2013 to December 2013 under the guidance and supervision of my unit chief **Prof. S.Tito, M.D.,** Professor of Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine – April 2014.**

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CONTENTS

Abstract

Introduction:

Alcoholism and related complications are more prevalent world wide. Alcohol use is associated with more than 60 medical conditions. Alcohol use disorders (AUD) generally confirmed by screening test based on questionnaires like AUDIT. Mean Corpuscular Volume also elevated in alcohol use disorders. Aim of this study is to evaluate usefulness of MCV as a marker of AUD.

Methods:

Totally 100 patients were evaluated. Patients who were admitted in medical wards of Rajiv Gandhi Government General Hospital, Chennai for found to having alcohol use disorders by AUDIT. Patients history were taken as per the questionnaire and patients were subjected to clinical examinations. Investigations like Complete Blood Count, Peripheral smear, Random Blood Sugar, Blood Urea, Serum Creatinine, Liver Function Test, Thyroid Stimulating Hormone Assay (TSH), USG abdomen also done and results were analysed.

Results:

Out of 100 patients with AUD, 61 patients had elevated MCV of >100 with statistically significant p value 0.028. 62 patients had elevated AST/ALT ratio of >1. To confirm this findings, large community based trails needed in persons with AUD without other medical comorbidity.

Key words:

AUDIT, AUD, MCV

INTRODUCTION

The intake of more than 3 standard drinks per day on continuously enhances the possibility of malignancy and vascular disease, and alcohol consumption in excess decrease the life span by about 10 years.

Alcohol distributes throughout the body, affecting almost all organs and altering nearly every neuro chemical process in the brain. Alcohol is likely to exacerbate most medical conditions, temporarily mimic many medical and psychiatric conditions.

In Western countries 80 percent of adult populations have drunk alcohol, and two-thirds have been drunk in the last year, the lifetime risk for serious, recurring alcohol problems is almost 20% for men and 10% for women, irrespective of a person's educational status or income.

Global scenario

Over all 3.5% of worldwide prevalence was caused by drinking alcohol, causing as much mortality and morbidity compared to tobacco and high blood pressure[3,4]. Alcohol consumption is causally related to more than 60 clinical conditions[5].

There is a rapid increase in per capita consumption of alcohol between1980and 2000 by over 50%. The fastest growth has been in the Asian countries[6.]

45% of total recorded alcohol is consumed in the form of spirits. 36% from beer, remaining contributed by wine and other beverages. Overall high alcohol consumption noted in the Russian Federation countries.

Trends in adult per capita consumption

Five year trend in alcohol consumption from 2001 to 2005 shows that there was a increase in alcohol consumption in developing countries. There was 68.3% increase in South East Asian countries and increase by 25.3% in African countries. The world wide increase in alcohol consumption noted was 23.5%.

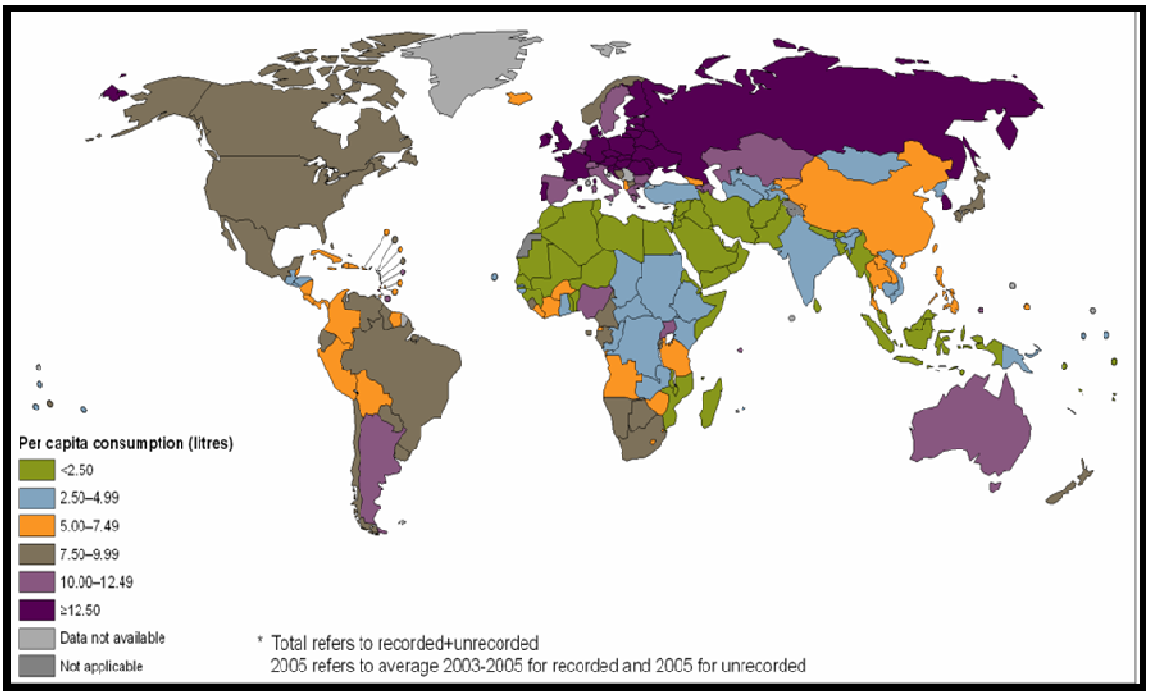
Alcohol consumption among young people

Global survey on health and alcohol conducted by WHO in 2008 shows that there was 71% increase in "underage drinking". Among 18 – 25 year old, there was 80% increase in alcohol consumption.

Alcohol rated mortality

42% alcohol attributable deaths were due to injuries. It was most commonly occurred during alcohol intoxication. Alcohol attributable mortality and disease burden per pure litre of alcohol consumption was more for underdeveloped and developing countries.

Figure 1 : Consumption of Alcohol in litres per capita – 2005



Indian scenario

The per capita intake of ethanol by an Indian adult aged more than 15 years **augmented by more than 100%between two decades**[7]. The drinking pattern also altered from occasional use to frequent use. Recently, these expansions have raised up worries about the medical and the communal significances of too much alcohol intake[8].

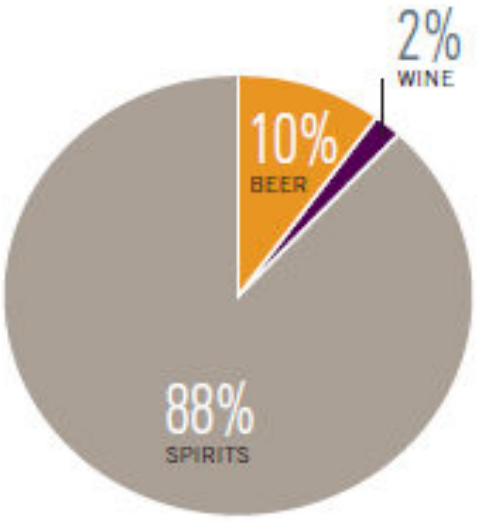
As per Government statistics, 21% of adult men and 2% adult women consumes alcohol. Among them, 20% were alcohol dependent who require management[35].

"Average age of initiation" of alcohol dropped from 19 years to13 years in last 20 years[35]. The fraction of people taking below 21 years has enlarged from 2% to 14% in the last 15 years.

More than 20% of hospital admissions, 18% of psychiatric emergencies and 60% of all injuries, 20% brain injuries who were attending hospital casualty rooms were related to alcohol[35]. Alcohol contributes to domestic violence. One third of domestic violence were related to alcohol intake.

Indians preferred to drink distilled alcohol more than wine and beer which contains higher level of pure alcohol (30 – 50%) leads to more complications.

Beverage preference in India



AIMS AND OBJECTIVES

- To evaluate the usefulness of mean corpuscular volume to identify alcohol use disorder in comparison with AUDIT (Alcohol Use Disorders Identification Test) screening test.

REVIEW OF LITERATURE

Alcohol not only affects the person who consumes it, also affects his or her family and society. Alcoholism mainly affects the persons of productive age group, it also related to drug abused, high risk sexual behavior, anti social activities. It affects the person irrespective of education and income. Alcoholism related complications are major threat to our country. Changing drinking pattern from occasional to social and acceptance of a drinking as a normal phenomenon, young drinkers all are major concern to our society.

Prevalence of alcoholism in rural Tamil Nadu shows that prevalence of alcohol use in adult male is 16.8% and in female it is 1.3%[42].

Types of Alcoholic Beverages

There are a more varieties of alcoholic drinks.

A. Malted liquors Acquired by fermentation of cereals; which are undistilled — alcohol content in them lies between 3-6% e.g. Beers, Stout. Now beers with the strength up to 10%) are also produced.

B. Wines Manufactured by fermentation of sugars existing mainly fruits like grapes and fruits. Wines are undistilled. When the amount of alcohol is 9-12%, they are called Light wines e.g., Cider is 9–12%, alcohol content cannot surpass 15%. When the distilled alcohol is added Fortified wines are made e.g., Sherry (alcohol 16–22%):

When bottled before fermentation of wines Champagne (12–16% alcohol) are made. Dry wine made from fermentation all sugars present in the wine and sweet wine made if when little sugar present.

C. Spirits These are produced by distillation after fermentation e.g. Rum, Brandy, Vodka and others. The amount of alcohol present in them is between 40–55%, in our country (and almost worldwide) 42.8% v/v or 37% w/w is standard accepted percentage worldwide for common brands.

The taste, aroma depends not only on alcohol content but on the availability of excess ethers, amount of aldehydes, esters, polymers, and volatile oils; most of them are produced during 'maturation' of the alcohol.

Other forms of alcohol

1. Dehydration alcohol produces- Absolute alcohol 99% w/w ethanol
2. From molasses by distillation produces Rectified spirit 90% w/w ethanol
3. Proof spirit made when whisky is drizzled on gun powder and burned and it blowup, then it was branded to be of 'proof strength'. If water is mixed to it, gun powder will not ignite.

Standard drinks.

In worldwide varies countries, health educators and investigators apply varies definitions of one standard drink because of differences in the usual serving amounts in that nation. For e.g., 1 unit of drink in Canada:

13.6 g of pure alcohol. 1 standard drink in the England: 8 g 1 s drink in the America: 14 g one standard drink in Australian continent: the AUDIT, Questions 2 and 3 accept that a "**standard drink equivalent is 10 grams"** of alcohol. The recommended low-risk consumption level set in the brief intervention manual and used in the WHO study interventions is not excess than twenty grams of alcohol per day, two days abstinence.

Calculation for the amount of Alcohol in a beverage

Amount of alcohol present in a drink depends on the strength of the drink and the content of the container. There are large differences in the strengths of alcoholic drinks and the drink sizes in different nations. A WHO survey showed that beer contains between 2%and 5% pure alcohol 10.5%to 18.9% in wines. In distilled spirits it is varied between24.3% to 90%, and in cider it is from1.1% to 17%. Hence, it is important to adjust drinking amounts to what is most prevalent at the native level and to recognize approximately the amount pure alcohol the individual drinks per time and on average.

Additional concern is calculating the volume of alcohol contained in a standard drink is the conversion factor of ethanol. That permits you to transform any amount of alcohol into gms.

For each ml of ethanol, there are 0.79 gms of pure ethanol. For (e.g.) beer of (330 ml at 5% x(strength) 0.79 (conversion factor) = 13 gm ethanol one glass wine (140 ml) at 12% x 0.79 = 13.3 grammes of ethanol one shot spirits (40 ml) at 40% x 0.79 = 12.6 gms of ethanol.

METABOLISM OF ALCOHOL

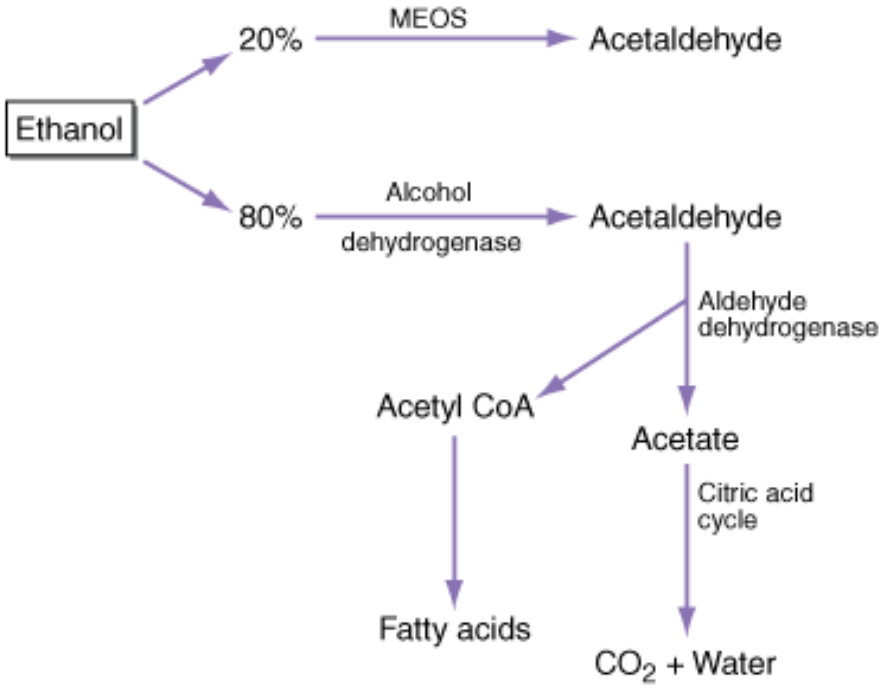
Alcohols are hydroxy product of aliphatic hydrocarbons. When complete, 'alcohol' means to ethyl alcohol or ethanol. Pharmacology of alcohol is significant for its presence in a drink (which have been used from the time olden days) and for liquor intoxication, rather than as a drug. Alcohol is mass-produced by fermentation of sugars: Zymase $C_6H_{12}O_6$ $2CO_2 + 2C_2H_5OH$ (in yeast) Fermentation continues till alcohol percentage reaches ~ 15%. Then the reaction is stopped by ethanol itself.

STARCH MALTOSE

. The fermentation cereal by yeast produces alcohol. Important source of available alcohols is mollases, a co product of sugar production. The most part of ethanol is metabolized mainly in the liver between 2% to 10% of ethanol is expelled straight through the lungs and also excreted through urine, or sweat. There are two pathways for alcohol metabolism one at cytosol and other one at microsomes of the smooth endoplasmic reticulum.

Acetaldehyde produced by action of cytosolic alcohol dehydrogenase (ADH). By aldehyde dehydrogenase (ALDH) rapidly converts in to other

metabolite in the cytoplasm as well mitochondria**.** Another pathway is in the microsome (the microsomal ethanol-oxidizing system or MEOS accountable for oxidative metabolism of ethanol).



Pharmacology and Nutritional Impact of Ethanol

Blood levels of alcohol are represented as mg or gm of ethanol per/dl. Alcoholic beverages also contains additional components known as congeners that affect taste of the drink and might contribute to adverse reactions on the body. Congeners include methanol, butanol, acetaldehyde, histamine, tannins, iron, and lead.

The higher the alcoholic content of the beverage, higher is the blood alcohol concentration (BAC). Alcohol is absorbed largely from the initial parts of the small intestine, although moderate amounts are absorbed from the stomach and proximal large bowl, and little amounts from the mucous

membranes of the mouth and oesophagus factors affecting blood alcohol concentration.

Drinking on an empty stomach, gulping drinks, mixing carbonated drinks, such as soda along with the alcohol, all enhance absorption.

Presence of fats, proteins and carbohydrates in the diet, presence of congeners can slow down absorption. Congeners found in alcoholic beverages, especially illicitly brewed alcohol (such as methanol, butanol, aldehydes, esters, histamines, phenols, tannins and heavy metals such as iron, lead and cobalt) can contribute to organ damage.

Blood alcohol peaks 30 to 60 minutes after consumption on an empty stomach. Women having higher fat content than men so they achieve higher blood alcohol for the same dose of alcohol.

INTERACTIONS

1. Alcohol synergises with anxiolytics, antidepressants, antihistaminics, hypnotics, opioids →marked CNS depression with defective motor function can occur: Probabilities of accidents in and injuries elevated many fold.

2. Persons taking anti diabetic drugs like sulfonylureas especially (chlor probamide not available in India), certain antibiotics like cephalosporins (cefoperazone, moxalactam, cefamandole) and

metronidazole have developed annormal, somewhat disulfiram-like adverse reactions when they drink alcohol.

3. Acute excess alcohol consumption inhibits, while prolnged intake induces metabolism of many drugs like anti diabetic drug tolbutamide, and anti epileptics like phenytoin and many other drugs
4. Interaction with insulin and sulfonylureas - these drugs are known to cause hypoglycemia by excess insulin. Alcohol reduces hepatic glycogen content when the alcoholics takes the medication risk of hypoglycemia increased many fold.
5. Alcoholics more prone to develop alcoholic gastritis and upper gastrointestinal bleeding when consumed in excess amount. NSAIDS like diclofenac which was commonly used as pain killer and antiplatelet like aspirin and steroids increases the risk of bleeding.
6. Paracetamol is known to cause liver damage. Alcoholics have reduced glutathione reserve so they more prone for paracetamol induced liver toxicity. However in patient with cirrhosis up to two gram of paracetamol can be given if medical condition needed it.

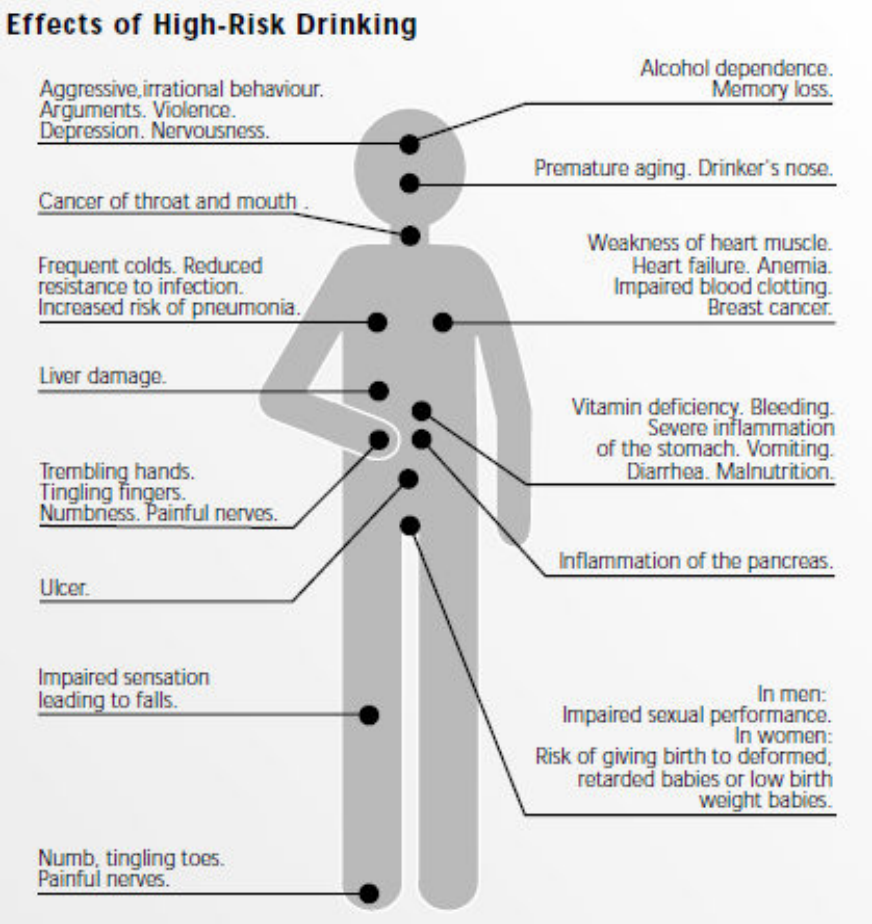
Tolerance to alcohol

Can occur due to three different mechanism:

- ❖ metabolic or pharmacokinetic tolerance occurs, after 1–2 weeks of daily alcohol consumption, it is due to accelerated in the rate of ethanol metabolism in the hepatic tissue. This change goes almost as quickly as it progresses.
- ❖ Cellular or pharmacodynamics tolerance occurs due to neurochemical changes that relatively maintains normal physiologic functioning inspite of the presence of alcohol. When three is decreases in blood levels leads on to development of with drawl symptoms.
- ❖ In third type of tolerance (learned or behavioural tolerance). Individuals function better than expected under influence of the alcohol.

The Effects of Ethanol in the varies body Systems[44]

Alcohol affects almost all the organ systems in the body. It associated with around 60 known medical conditions.



Nervous System

Roughly 35% of alcoholics had an episode of temporary anterograde memory loss, in which the individual forgets all or part of events that happened during a drinking period that is called as blackout.

Disturbed sleep is an another common problem, alcohol may help a person to fall asleep initially, but it disrupts sleep all over the rest of the sleep by altering the varies stages of sleep and hours spent in rapid eye movement (REM) and deep sleep is reduced excess snoring and exacerbate sleep apnoea occurs in alcoholics due to relaxation of muscles in the

pharynx of alcoholics may also experiences disturbing dreams. Sleep related problems are more marked in alcoholics, and their persistence may lead on to relapse.

Impaired judgment and coordination are common consequence of alcohol use, increases the risk of accidents and injury. In the United States, excessive alcohol consumption can also be associated with headache, polydipsia, nausea, vomiting, and excessive tiredness the following day, it is called as hangover syndrome .that is responsible for much missed time and temporary cognitive impairment at workplace.

Chronic excess alcohol doses cause peripheral neuropathy in 10% of alcoholic patients clinically presented as bilateral limb numbness which is more pronounced distally, tingling, and paraesthesia. Approximately 1% of chronic alcoholics develop cerebellar degeneration or atrophy. This syndrome characterised by advanced imbalance in stance and gait often associated nystagmus. MRI brain studies shows atrophic changes in the cerebellar vermis.

Wernicke's (ophthalmoparesis, ataxia, and encephalopathy) and Korsakoff's (retrograde and anterograde amnesia) syndromes are rare occurs 1 in 500 alcoholics. These syndromes occur as the result of reduced levels of thiamine.

Decreased Brain volume, evident as ventricular enlargement and widened cortical sulci on neuro imaging, occurs in fifty% of chronic alcohol drinkers; these changes are usually normalise if abstinence is continued for years.

Psychiatric problems

As for Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) two-thirds of alcohol-dependent individuals meet the criteria for a psychiatric syndrome.

50% of these relate to a underlying antisocial personality manifesting as impulsive behavior and loss inhibition that contribute to both alcohol and drug dependence.

Others have psychiatric syndromes have psychiatric conditions such as schizophrenia or mania or depression disease and anxiety disorders such as panic disorder. Many psychiatric syndromes can be seen in the short term during excess alcohol intake and followed by withdrawal.

Alcohol-induced mood disorder

These alcohol-induced mood disorder include an marked low mood lasting for days to weeks in the midst of excess drinking documented in 40% of alcoholics, it will take several weeks to resolving of symptoms after stopping alcohol.

Alcohol-induced anxiety disorder

Profound anxiety occurs in 10–30% of alcohol drinkers temporarily. Usually starts at the time alcohol withdrawal, which may persist for a month or more after stopping of alcohol ingestion.

alcohol-induced psychotic disorder

Auditory hallucinations and/or paranoid delusions in a person who is alert and oriented, seen in 3–5% of alcoholics. Alcohol-induced conditions are temporary and usually does not need long-term pharmaco therapy, any patient who has above mentioned psychiatric symptoms history of excessive alcohol use is an important part of the workup .

The Gastrointestinal System

Esophagus and Stomach

Excess Alcohol ingestion can cause marked inflammation of the upper gastero intestional tract mainly esophagus and stomach causing epigastric discomfort and upper gastrointestinal bleeding. Haemorrhagic gastritis is Commonly caused by excess Alcohol consumption. Forceful vomiting after alcohol ingestion can produce severe bleeding through a Mallory-Weiss tear, a longitudinal tear in the mucosal layer of gastroesophageal junction.

Pancreas

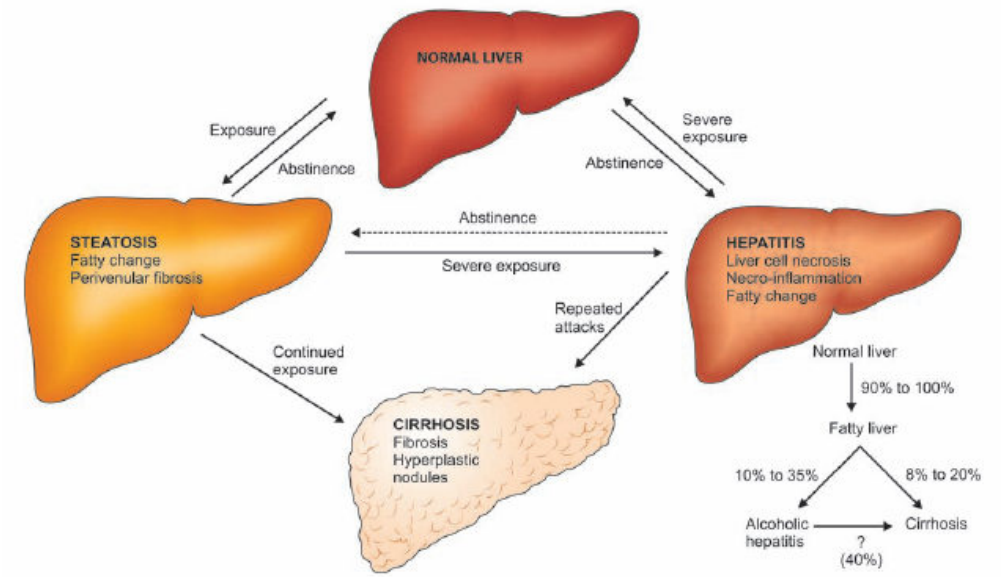
Alcoholic develops acute pancreatitis almost three times higher than the general population, contributed to 10% or more of the total cases.

Liver

Long term alcohol intake can leads on to spectrum of liver pathology that varies from mild fatty liver to cirrhosis and HCC(hepatocellular carcinoma). Fat accumulation in liver cells is seen in 90% of alcoholic fatty change is most predictable and earliest response to alcohol ingestion. impaired glucose production ,in the liver, resultant in a fall in the quantity of glucose made from glycogen, reduced oxidation of fatty acids and increased lactate production, in alcoholics leads onto an increase in fat accumulation in hepatic cells causes fatty liver.

Risk factors for development of alcohol related liver disease includes gender, associated hepatitis c infection, poor nutrition, heritable and the Amount of alcohol ingested. Type of alcohol usually not related to alcoholic liver disease, female are more prone to develop with less quantity, high fat intake predispose and coffee intake has productive effect.

Spectrum of Alcoholic Liver Diseases



10% of people will develop cirrhosis if they continuous drink heavily. Patients with both micro- and macro vesicular pattern of steatosis having more risk of developing cirrhosis than patients with only the macro vesicular[2]. About 10to35% of heavy drinkers will develops alcoholic hepatitis it is a prerunner of cirrhosis associated with high short term mortality. Continued alcohol ingestion, causes (micro nodular, or Laennec's, cirrhosis) in 8% to 20% of excess alcohol consumper. Over time, and especially with long term abstinence from alcohol, this changes can evolves into (macro nodular cirrhosis).

In patients who have had complications of cirrhosis and who continue to take alcohol, 5 year survival rate reduced to50%.[1] In contrast, in those patients who are able to remain abstinent, the prognosis is significantly improved.

Alcoholics have an increased rate of hepatitis C infection, and excess alcohol consumption in the setting of hepatitis c infection more severe liver deterioration will occur.

Cancer

Alcohol promotes cancer by direct effects of alcohol and acetaldehyde. And another mechanism is by altered immune homeostasis. As low as 1.5 drinks per day increases risk of carcinoma breast in a female by1.4-fold. For both males and females, four drinks per day elevate the risk for oral and oesophageal malignancy threefold and rectal cancers 1.5 fold; seven to eight or more drinks per day increases approximately five times the risks for so many malignancies.

Hematopoietic System

Excess alcohol intake causes an increase in red blood cell size [mean corpuscular volume (MCV)], by affecting stem cells. If heavy drinking is associated with by folic acid deficiency, along with increased red blood cell size, hyper segmented neutrophils, reticulocytopenia, and a hyperplastic bone marrow will also present. If alcohol intake associated with malnutrition sideroblastic changes can also be observed. White blood cell production decreased in Chronic alcoholics. It also affect granulocyte mobility and adherence, and false-negative tuberculin skin test occurs

because of impaired delayed-hypersensitivity responses to antigens. Hepatitis and HIV infection risk is higher than in general population due to associated immune deficiency and altered liver function interfere with treatment. Mild thrombocytopenia common in alcoholics usually it resolves within a week after cessation of alcohol. When it does not resolve we have to rule out cirrhosis of liver or congestive splenomegaly.

Cardiovascular System

Acute alcohol consumption causes peripheral vasodilation and it decreases myocardial contractility, resulted in mild reduction in blood pressure and also compensatory rise in cardiac output. Cardiac oxygen consumption during exercise is higher after alcohol consumption. When the patient having underlying cardiac disease these changes can affect damaged heart.

There will be a dose-dependent increase in blood pressure will occurs, then the patient drinks of three or more drinks per day. Blood pressure will be normalized after cessation of alcohol. Thus, excess alcohol consumption is an important risk factor for mild to moderate hypertension. risk for coronary artery disease is six times higher for chronic excessive drinker, may related to increased low-density lipoprotein cholesterol. Alcohol directly affects cardiac muscle and causes cardio myopathy. Unexplained arrhythmias, cardiac failure, hypo contractility of cardiac

muscle, and dilation of both ventricle and atrium lead on mitral regurgitation and formation of mural thrombi all will occur because of excess alcohol. Holiday heart syndrome characterized by paroxysmal tachycardia in a structurally normal heart after excess alcohol consumption it is due to Atrial or ventricular arrhythmias. It normalize after cessation of alcohol

Genitourinary System

Excess alcohol consumption in teen age boys and girls can affect normal sexual development and reproductive onset. Although alcohol in moderation increases the sexual drive but it causes impaired erectile function in men. Even in the absence of hepatic impairment. Permanent loss of testicular volume with atrophy of the semniferous tubules, decreases in seminal volume as well as low sperm count will occur in chronic alcohol drinker.

The continuous ingestion of high doses of alcohol by the women who consumes large doses alcohol will develop amenorrhea, reduction in ovarian size, corpora lutea will become absent and they become infertile. When they become pregnant there is an increased risk of spontaneous abortion. Excessive drinking during ante natal period results in fetal alcohol syndrome (FAS), it is due to rapid transfer of alcohol and its product acetaldehyde, through placenta to the fetus. Milder form this condition is

called fetal alcohol spectrum disorder (FASD). Not even a little amount of alcohol is safe during pregnancy, there is no specific cut off for safe drinking during pregnancy period. Alcohol should be avoided completely.

Fetal alcohol syndrome (FAS) characterized by facial dimorphism such as microcephaly, epicanthal eye folds; poorly formed ears; small teeth with defective enamel; cardiac anomalies in the form of septal defects; an unusual palmar crease and restriction in joint movements; and with mental retardation.

Fetal alcohol spectrum disorder (FASD) described by decreased birth weight, a low IQ, and hyperactive behavior, and mild cognitive impairment.

Acute alcoholic myopathy can occurs alcoholics characterized by skeletal muscle weakness that partially improved after abstinence caused by osteoporosis and osteo necrosis occurs in chronic alcoholic due to alteration in the calcium metabolism. Effects of chronic excessive drinking on the skeletal system comprise of changes in calcium metabolism, and decreased bone mineral density, and reduced rate of growth in the epiphyses, leads on to augmented risk fractures and osteonecrosis of the head of femur.

Hormonal changes

In healthy individual transient hypoglycemia can occur following excessive alcohol consumption within 6–36 h, it is due to the actions of

alcohol on glucose formation by inhibiting gluconeogensis. This can result in transient abnormality in glucose tolerance tests (with a resulting wrong diagnosis of diabetes mellitus) until the alcoholic has abstained for minimum two weeks.

Alcohol ketoacidosis can occur because of reduction in fatty acid oxidation along with decreased intake or repeated vomiting can be wrongly diagnosed as diabetic ketoacidosis. It can be differentiated by normal to mildly elevated blood sugar usually less than 250 mgs /dl in alcoholic keto acidosis. And by clinical feature like parotid swelling.

Cortisol levels are elevated in alcoholics, it correlated with excessive alcohol consumption. Usually alcoholic are over hydrated because of inhibition of vasopressin secretion at increasing blood alcohol concentrations and increased secretion at falling blood alcohol concentrations. So when treating alcohol intoxication intravenous fluids are not nessary unless dehydration is present.

Serum triiodothyronine (T_3) level decrease more markedly in comparison with serum thyroxine (T4) which is decreased in modest amount. Hormonal problems should be reassessed as they may disappear after a month of alcohol abstinence.

Whenever a patient come for below mentioned problems, we have to think alcohol as attributing factor and further enquiries should be made.

- Adult onset seizure
- Young stroke
- Poorly controlled hypertension
- Young onset or poorly controlled diabetes
- Hepatitis, cirrhosis
- Peripheral neuropathy
- Pancreatitis
- Parotid gland enlargement
- Cancer
- Gastrointestinal symptoms of undermined cause
- Sleep disturbances
- Unexplained falls or accidents
- Poorly controlled gout
- Confusion or cognitive impairment
- Non-specific somatic symptoms
- Work-related problems such as absenteeism

Withdrawal Syndrome

A withdrawal state is regarded as by a group of symptoms, often specific to the drug used, which follows on complete or partial withdrawal of a drug, generally after recurrent and/or high-dose use. This, too, is a short-lasting syndrome with usual duration of few hours to few days.

Typically, the patient reports that the withdrawal symptoms are controlled by further substance use. The withdrawal state is further classified as:

i. uncomplicated

ii. with convulsions; and

iii. with delirium

Withdrawal Syndrome

The most common withdrawal syndrome is a hangover on the next morning. Mild tremors, nausea, vomiting, weakness, irritability, insomnia and anxiety are the other common withdrawal symptoms. Sometimes the withdrawal syndrome may be more severe, characterized by one of the following three disturbances: delirium tremens, alcoholic seizures and alcoholic hallucinosis. It is important to remember that alcohol withdrawal syndrome can be associated with marked morbidity as well as significant mortality, and it is important to treat it correctly.

Delirium tremens (DT) is the dangerous form of alcohol withdrawal syndrome. It comes about generally within 2-4 days of total or significant abstinence from excess alcohol drinking in about 5% of patients, as related to acute tremlousness which occurs in about 34%of patients.

The course is brief, with improvement occurring within 3-7 days. This is an acute organic brain syndrome (delirium) with following clinical features:

i. Impairment of consciousness with not oriented to time and place.
ii. Lack of attention and easy distractibility.
iii. Visual (and also auditory) hallucinations and illusions which are often vivid and very frightening.
iv. insects crawling sensation over the body may occur called as Tactile hallucinations
v. Autonomic disturbance in the form increased pulse rate, fever, elevated blood pressure, sweating and pupillary dilatation.
vi. Psychomotor agitation and ataxia.
vii. Insomnia, with a reversal of sleep-wake pattern
viii. Dehydration with electrolyte disturbance. Death can occur in 5-10% of patients with deliriumtremens and is often due to cardiovascular collapse, infection, hyperthermia or self-inflicted injury. At times, intercurrent medical illnesses such as pneumonia,

fractures, liver disease or pulmonary tuberculosis may complicate the clinical picture.

2. Alcoholic seizures ('rum fits')

Generalised tonic clonic seizures occur in about 10% of alcohol dependence patients, usually 12-48 hours after a heavy bout of drinking. Often these patients have been drinking alcohol in large amounts on a regular basis for many years. Multiple seizures (2-6 at one time) are more common than single seizures. Sometimes, status epileptics may be precipitated. In about 30% of the cases, delirium tremens follows.

3. Alcoholic hallucinosis

Alcoholic hallucinosis is characterized by the presence of hallucinations (usually auditory) during partial or complete abstinence, following regular alcohol intake.

It occurs in about 2% of patients. These hallucinations persist after the withdrawal syndrome is over, and classically occur in clear consciousness.

Usually recovery occurs within one month and the duration is very rarely more than six month.

Alcohol dependence - biopsychosocial disorder

Biological Vulnerability

There have been many studies to support the heritable nature of alcoholism including family, twin and adoption studies, which indicate a four-fold increase in the risk for developing alcoholism in children of alcoholics. Studies suggest that the inherited risk for the development of alcohol dependence is 50% to 60%.

Neurotransmitter Changes after alcohol use Different neurotransmitters are also known to alter alcohol consumption behaviour. Dopamine may contribute to the motivation and reinforcement of alcohol consumption.

Serotonin levels appear to be lower in the brains of alcohol dependents compared to non alcoholics. It may promote alcohol's intoxicating and rewarding effects.

Glutamate receptors in the brain are inhibited by alcohol land this may account in part for the cognitive dysfunction that occurs with alcohol intake. Mediate the sedative effects of alcohol.

Alcohol-induced modifications in GABA receptor function may leads on to alcohol dependence and tolerance, and abnormalities in the GABA system may predispose to alcoholism. In addition, various neuromodulators affected by alcohol include endogenous opioid peptides (which may mediate alcohol

reinforcement and excessive alcohol consumption), adenosine(which may mediate many of alcohol's acute and chronic effects on the CNS such as incoordination, intoxication and sedation)

Psychosocial Theories

Social factors, such as broken families, single parent families, parental 'models', peer pressure, availability of alcohol and cultural sanction of drinking, all contribute to the progress of alcohol abuse and dependence.

Psychological vulnerability, including stress full life situations, low self-esteem, poor coping and low communication skills may predispose to alcoholism. Alcoholism has also been described as a learnt behaviour, with the temporary relief from anxiety with alcohol acting as a reinforce,perpetuating alcohol use.

Thus, alcohol dependence may be aptly described as a **biopsychosocial disorder**, with a complex interplay of environmental, genetic and psychosocial factors.

Etiological factors in alcohol abuse

1. Biological Factors
i. Genetic vulnerability (family history of substance
use disorder; for example in type II alcoholism)
ii. Co-morbid psychiatric disorder or personality
disorder
iii. Co-morbid medical disorders
iv. Reinforcing effects of drugs (explains continuation
of drug use)
v. Withdrawal effects and craving (explains continuation
of drug use)
vi. Biochemical factors (for example, role of
dopamine and norepinephrine in cocaine,
ethanol and opioid dependence)
2. Psychological Factors
i. Curiosity; need for novelty seeking
ii. General rebelliousness and social non-conformity
iii. Early initiation of alcohol and tobacco
iv. Poor impulse control
v. Sensation-seeking (high)
vi. Low self-esteem (anomie)
vii. Concerns regarding personal autonomy
viii. Poor stress management skills
ix. Childhood trauma or loss
x. Relief from fatigue and/or boredom
xi. Escape from reality
xii. Lack of interest in conventional goals
xiii. Psychological distress
3.Social Factors
i. Peer pressure (often more important than
parental factors)
ii. Modelling (imitating behaviour of important
others)
iii. Ease of availability of alcohol and drugs
iv. Strictness of drug law enforcement
v. Intrafamilial confl icts
vi. Religious reasons
vii. Poor social/familial support
viii. ‘Perceived distance’ within the family
ix. Permissive social attitudes
x. Rapid urbanisation

Management of alcohol related problems

Early intervention

Studies have shown that early, brief intervention can be very useful. A trial period of reduction of drinking or abstinence and review of the presenting problems can be suggested.

Abstinence must be strongly advised in the following most vulnerable groups such as patients with a serious problem or a potential risk of serious problems with alcohol (dependent use family history of dependence, physical illness such as liver dysfunction, cognitive impairment, psychiatric illness, and during pregnancy, serious social crises, e.g. threat of job loss or separation of family).

Clear statement of the physician's medical concern, involvement of the care givers and significant others in counseling and a joint agreement with the patient regarding the treatment goal have been shown to have significant results in early stage drinkers.

All alcohol dependents should be motivated for longer and more intensive treatment programmes. Monitoring health and drinking state at every follow-up is crucial to maintain the benefits of treatment.

For dependence

For patients with dependence, treatment consists of managing withdrawal-related syndromes and long-term treatment. Withdrawal, it, is best treated

pharmacologically, whether dependence is moderate or severe to avoid neurotoxicity associated with abrupt withdrawal of alcohol from the brain.

Outcome will be similar whether treated as outpatient (or) inpatient care is preferred in the following situations serious physical or psychiatric illness, at risk for seizures or delirium, failed-outpatient detoxification, poor social supports and severe alcohol craving.

management of the dependent alcoholic include:

Thorough clinical examination and investigations for assessment of nutritional impairment, electrolyte imbalance, cardiac,hepatic, gastrointestinal and neurological impairment.

Adequate nutrition and rest. Oral or parenteral B-complex can be provided, including 50 to 100 mg thiamine, for 7 to 10 days. Symptomatic treatment of underlying problems such as gastritis, diarrhea or dehydration.

Detoxification

Drugs commonly used to control withdrawal symptoms are the long-acting benzodiazepines (diazepam 5 to 10 mg or chlordiazepoxide 25 to 50 mg 6 hourly) to ameliorate the withdrawal symptoms.

In the presence of liver dysfunction Short-acting benzodiazepines (lorazepam 1 to 2 mg or oxazepam 15 to 30 mg repeated four hourly) are preferred .

The drug dose can be reduced by 20% on subsequent days and gradually tapered over a week to ten days. Other drugs that are tried for detoxification include anticonvulsants, such as carbamazepine, phenytoin, gabapentin, alpha-adrenergic agents such as clonidine, and beta-blockers such as propranolol

Delirium tremens can be mainly managed with supportive care, correction of fluid and electrolyte imbalance, antibiotics for control of infection, high doses of benzodiazepines along with parenteral B-complex.

Antipsychotics, such as haloperidol in low doses (2.5 to 5 mg) may be helpful in controlling agitation, but must be used carefully as they may reduce seizure threshold. Alcohol withdrawal seizures do not need long-term anticonvulsant treatment.

Following detoxification, the patient's motivation is strengthened through individual, group and family counselling.

Following are included in the counseling educating about alcoholism, interventions to deal with interpersonal, marital, sexual problems, vocational counselling, helping the patient develop better skills of communication, assertiveness (e.g. handling peer pressure), problem solving and dealing with stress. The family is supported and helped to provide a supportive environment to the patient to facilitate recovery.

For women alcoholics, gender-sensitive treatment programmes addressing psychological and family issues more intensively, need to be provided. Self-help groups, such as the alcoholic anonymous (AA) and Al-Anon, for families are perceived as useful by many patients. Treatment of less severely dependent alcoholics in rural areas has also been successfully carried out through camps.

Pharmacotherapy for long-term treatment

Treatment Psychological and medical supportive measures are required throughout withdrawal many CNS sedatives like barbiturates, phenothiazines, have been used as replacement Therapy in the past (to reduce withdrawal syndrome) but benzodiazepines like(chordiazepoxide, diazepam) are the favored drugs now. These have a extended duration of action and can be slowly withdrawn later.

Naltrexone: Numerous studies have established involvement of opioid system in the enjoyable reinforcing effects of alcohol probably by diminishing dopamine mediated reward function. Studies between post-alcohol dependent have shown that the long acting opioid antagonist naltrexone helps prevent relapse of alcoholism. It reduced alcohol craving, number of drinking days and chances of continued excess drinking. Naltrexone is accepted by US-FDA for use as adjuvant in comprehensive management programmes for alcohol dependent patients. In our country majority of deaddiction centres, use naltrexone after the individual has undergone withdrawal and is motivated.

Acamprostate

It is a weak NMDA-receptor antagonist with uncertain GABAA receptor agonistic action .that is being tried in European countries for maintenance therapy of alcohol cessation. In combination with communal and motivational treatment, it has been found to decrease revert of the drinking behavior. The usefulness of acomprostate in this regard is valued similar to naltrexone.

Ondansetron a 5-HT3 antagonist and the anti seizure drug topiramate have also shown some usefulness in the management of alcoholism.

Aldehyde dehydrogenase inhibitors

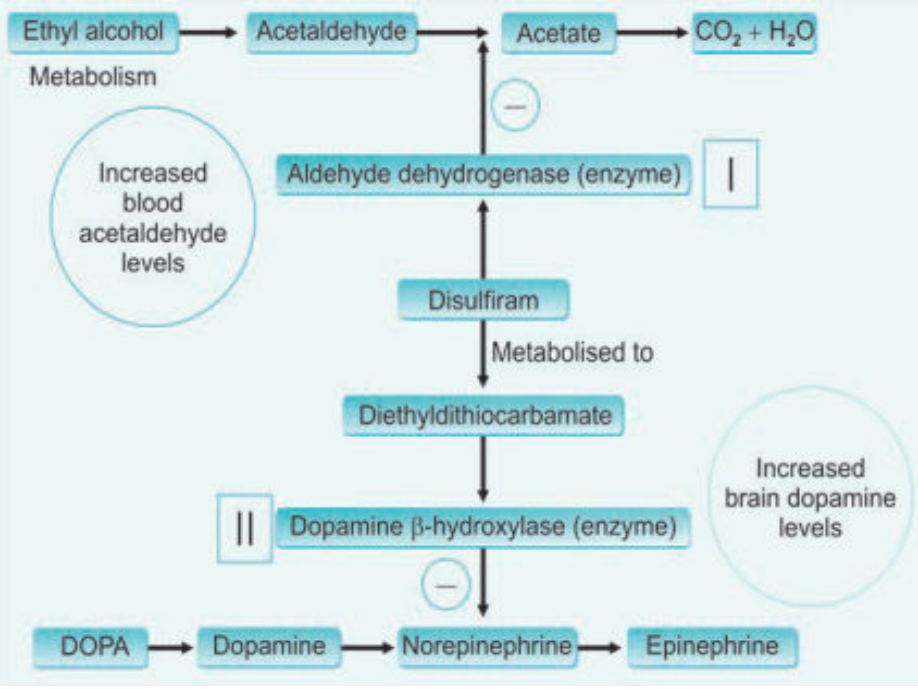
Disulfiram

The enzyme aldehyde dehydrogenase inhibited by disulfiram most likely after change into active metabolites. When alcohol is consumped after taking disulfiram, the level of acetaldehyde in tissues and blood increases and a lot of extremely disturbing symptoms (aldehyde syndrome) are formed quickly. Symptoms ar— hot flushing, burning sensation, severe headache, increased sweating, uneasiness, chest tightness, dizziness, vomiting, visual problems, alteration in the mentation, postural fall and circulatory collapse. Spell of the syndrome (1–4 hours) be influenced by on the amount of alcohol taken. Because of risk of adverse reaction, disulfiram is not frequently used. Disulfiram has been given as an aversion method in chronic alcoholics who are encouraged and genuinely desire to leave the alcohol intake. After stopping alcohol after 8- 12 hours preferably after overnight, disulfiram is given 1 g on day one, 0.75 g on 2

day two , 0.5 g on day 3 and 0.25 g afterwards. Aldehyde syndrome to alcohol develops after 2–3 hours of first dose, touches its peak at ~12 hours and lasts for 7–14days after discontinuing it, because inhibition of aldehyde dehydrogenase with disulfiram is irreversible: synthesis of new enzyme is necessary for return of activity. Thus, the patient resolve not to consume alcohol is reinforced by the disturbing symptoms that occur if he takes a little bit of alcohol it should not begiven to the individuals who are physically dependent on alcohol.

Side effects of disulfiram (as such) are not frequent; include skin reactions, metallic taste, nervousness, easy fatigue ability and abdominal discomfort. It inhibits a number of other enzymes as well including alcohol dehydrogenase, dopamine hydroxylase.

Mechanism of action of disulfiram



Monitoring and Follow-Up

Alcoholism, like other chronic illnesses such as hypertension or diabetes has a chronic, relapsing course. While 50% to 60% of patients significantly reduce their drinking or abstain for 6 months, relapse is frequent. Follow-up, therefore is mandatory and is very useful in bringing the patient back into treatment. In addition, as end stage problems are more resistant to treatment, the focus needs to shift to early detection and intervention for alcohol-related problems.

Alcohol intoxication Alcohol intoxication

Acute intoxication is a temporary illness resulting after administration of alcohol resulting in disturbances in level of consciousness, cognition, perception, affect or behavior, or other psychophysiological functions and responses. This is usually associated with high blood levels of the alcohol. However, in certain cases where the threshold is low (due to a serious medical illness such as chronic renal failure or idiosyncratic sensitivity) even low dose may lead to intoxication. The severity of intoxication reduces with time, and effects in due course vanish in the absence of further use of the alcohol.

The recovery is complete, excepting where tissue damage or another problem has arisen.

The following codes may be used to indicate whether the acute intoxication was associated with any complications:

i. uncomplicated
ii. with trauma or other bodily injury;
iii. with other health complications (such as blood vomiting, aspiration of vomitus);
iv. with delirium;
v. with perceptual distortions;
vi. with loss of consciousness;
vii. with seizure; and
viii. pathological intoxication (only for alcohol).

The signs of alcoholic intoxication clinically resembles other central nervous system depressant overdose .excessive drowsiness, errors of commission, psychomotor dysfunction, disinhibition, slurring of speech , ataxia, and nystagmus. Symptoms depends on blood level of alcohol.

Intoxication as manifested by ataxia, dysarthria, and nausea and vomiting indicates a blood level 150 mg/dL, blood levels range between350 to900 mg/dL is highly lethal. Marked by respiratory depression, stupor, seizures, shock syndrome, coma, and eventually death.

The first priority in treating alcohol intoxication is to maintain vital signs and manage respiratory depression, managing cardiac arrhythmia. If Aggressive behaviour present initially treated by reassurance if needed some force if it is not controllable then pharmacotheraphy should be tried with short acting benzodiazepine or anti psychotics like haloperidol.

Effects of Blood Alcohol Levels in the Absence of Tolerance

Blood Level, g/dL	Usual Effect
0.02	Decreased inhibitions, a slight feeling of intoxication
0.08	Decrease in complex cognitive functions and motor performance
0.20	Obvious slurred speech, motor incoordination, irritability, and poor judgment
0.30	Light coma and depressed vital signs
0.40	Death

Guidelines for safe drinking

Doctors are frequently requested to guide on safe ways of drinking. Different official organizations, doctor organizations and alcoholism specialists have put forth guiding principle in this regard, but they are not identical. The following may be concluded.

• On an average 1–2 drinks per day is generally safe.
• Not more than three drinks on any one time
• Consumption of >3 drinks per day is associated with recognized adverse health effects.
• Do not drive or engage in hazardous activities after alcohol.
• Do not drink if an interacting drug has been taken.
• Subjects with any contraindication should not take alcohol.
• Safe limits are somewhat lesser for females.

Alcohol Use disorders

It consists of:

- ❖ alcohol dependence
- ❖ Alcohol abuse or harmful use

Alcohol dependence was previously called as alcoholism. This term much like 'addiction' has been dropped due to its derogatory meaning. According to Jellinek, there are five 'species' of alcohol dependence (alcoholism) on the basis of the patterns of use (and not on the basis of severity).

A. Alpha (α)

i. Excessive and inappropriate drinking to relieve physical and/or emotional pain.

ii. No loss of control.

iii. Ability to abstain present.

B. Beta (β)

i. Excessive and inappropriate drinking.

ii. Physical complications (e.g. cirrhosis, gastritis and neuritis) due to cultural drinking patterns and poor nutrition.

iii. No dependence.

C. Gamma (γ); also called as malignant alcoholism

i. Progressive course.

ii. Physical dependence with tolerance and withdrawal symptoms.

iii. Psychological dependence, with inability to control drinking

D. Delta (δ)

i. Inability to abstain.

ii. Tolerance.

iii. Withdrawal symptoms.

iv. The amount of alcohol consumed can be controlled.

v. Social disruption is minimal

E. Epsilon (ε)

i. Dipsomania (compulsive-drinking).

ii. Spree-drinking.

Earlier, it was believed that γ-alcoholism was more common in America, while δ-alcoholism was commoner in the wine-drinking countries such as France. At present the existence of this pattern of distribution is doubted and its inclusion in this book is mainly for historical reasons.

Cloninger has classified alcoholism into two types, on the basis of the relative importance of genetic and environ mental factors.

classification of alcoholism

Factors	*Type I*	*Type II*
Synonym	Milieu-limited	Male-limited
Gender	Both sexes	Mostly in males
Age of onset	> 25 years	< 25 years
Aetiological factors	Genetic factors important; strong *environmental influences* are contributory	*Heritable*; environmental influences are limited
Family history	May be positive	Parental alcoholism and antisocial behaviour usually present
Loss of control	Present	No loss of control
Other features	Psychological dependence; and guilt present	Drinking followed by aggressive behaviour; spontaneous alcohol seeking
Pre-morbid personality traits	Harm avoidance; high reward dependence	Novelty-seeking

Alcohol dependence is more common in men, and has an onset in later part of second or early part of third decade. The course is generally insidious. There is often an connected abuse or dependence of other drugs. If the onset occurs late in life, especially after forty years of age, an underlying mood disorder should be looked for.

DSM IV give the following definition:

To diagnose alcohol dependence three out of seven symptoms should be present within 12 months period.

ALCOHOL DEPENDENCE

(A) A maladaptive pattern of drinking, leading to clinically significant impairment or distress, as manifested by three or more of the following occurring at any time in the same 12-month period:

Need for markedly increased amounts of alcohol to achieve intoxication or desired effect; or markedly diminished effect with continued use of the same amount of alcohol.

The characteristic withdrawal syndrome for alcohol; or drinking (or using a closely related substance) to relieve or avoid withdrawal symptoms.

Drinking in larger amounts or over a longer period than intended.

Persistent desire or one or more unsuccessful efforts to cut down or control drinking.

Important social, occupational, or recreational activities given up or reduced because of drinking.

A great deal of time spent in activities necessary to obtain, to use, or to recover from the effects of drinking.

Continued drinking despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to be caused or exacerbated by drinking.

(B) No duration criterion separately specified, but several dependence criteria must occur repeatedly as specified by duration qualifiers associated with criteria (e.g., “persistent,” “continued”).

Alcohol abuse: To diagnose alcohol abuse, only one symptom from the below mentioned is enough over a period of one year

ALCOHOL ABUSE

(A) A maladaptive pattern of drinking, leading to clinically significant impairment or distress, as manifested by at least one of the following occurring within a 12-month period:

Recurrent use of alcohol resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences or poor work performance related to alcohol use; alcohol-related absences, suspensions, or expulsions from school; neglect of children or household)

Recurrent alcohol use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by alcohol use)

Recurrent alcohol-related legal problems (e.g., arrests for alcohol-related disorderly conduct)

Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol (e.g., arguments with spouse about consequences of intoxication).

(B) Never met criteria for alcohol dependence.

There are several questionnaires used to find out alcohol used disorders. Commonly used tests are:

- CAGE
- MAST
- AUDIT

CAGE: It is popular in the primary care settings because of its simple nature. It can find out AUD. If it is positive, further evaluation is needed.

CAGE Screen For Alcohol Abuse.

C =	"Have you ever felt you should **C**ut down on your drinking?"
A =	"Have people **A**nnoyed you by criticizing your drinking?"
G =	"Have you ever felt bad or **G**uilty about your drinking?"
E =	"Have you ever had a drink as an **E**ye-opener first thing in the morning to steady your nerves or help a hangover?"

Yes to two or more: probable alcohol abuse

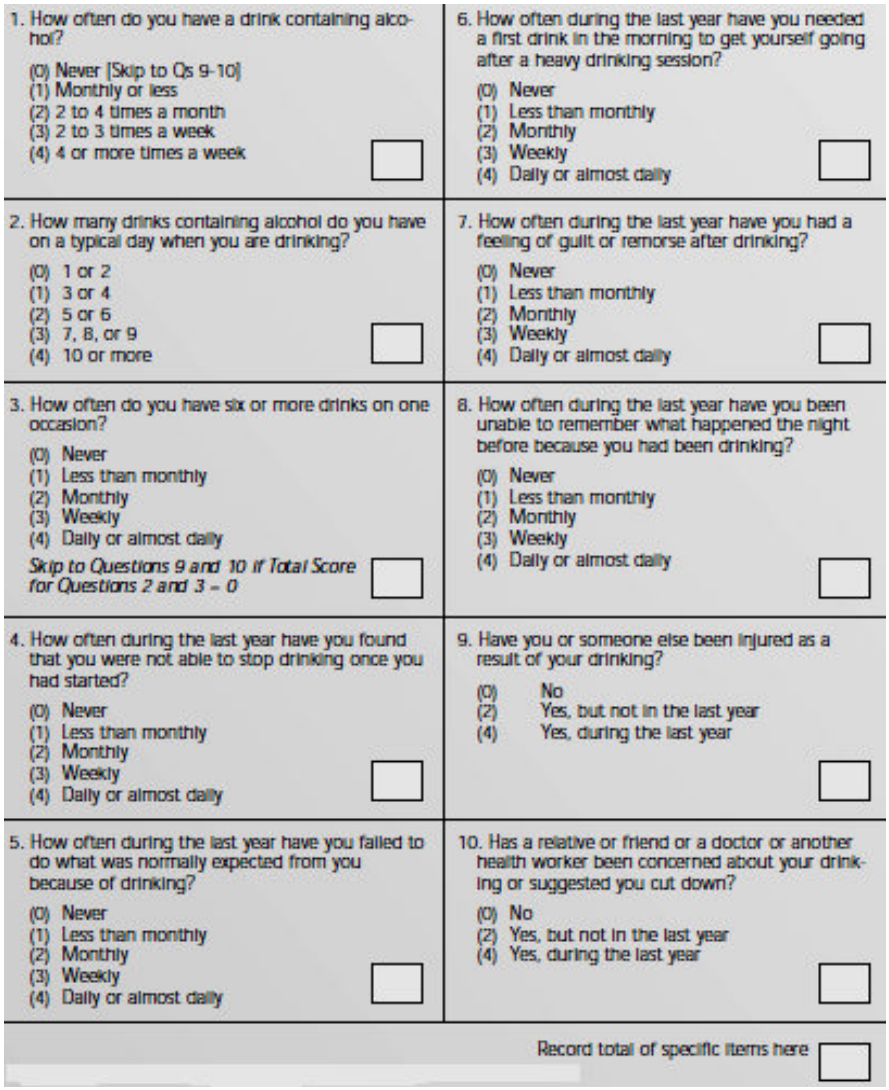
MAST (Michigan Alcoholism Screening Test)

It is an age old screening test to identify alcohol related problems and drinking behaviour. It contains 25 questions. There are several modifications came in MAST.

AUDIT (Alcohol use disorder identification test)[52]

It was developed under the guidance of World Health Organization. It has 10 questions. It has a ability not only to find out alcohol dependence, it can identify

patients with drinking without alcohol dependence. It validated internationally in all ethnic groups. It is the only screening test specifically developed for worldwide use.



1. How often do you have a drink containing alcohol?
(0) Never [Skip to Qs 9-10]
(1) Monthly or less
(2) 2 to 4 times a month
(3) 2 to 3 times a week
(4) 4 or more times a week

2. How many drinks containing alcohol do you have on a typical day when you are drinking?
(0) 1 or 2
(1) 3 or 4
(2) 5 or 6
(3) 7, 8, or 9
(4) 10 or more

3. How often do you have six or more drinks on one occasion?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily
Skip to Questions 9 and 10 if Total Score for Questions 2 and 3 = 0

4. How often during the last year have you found that you were not able to stop drinking once you had started?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

5. How often during the last year have you failed to do what was normally expected from you because of drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

7. How often during the last year have you had a feeling of guilt or remorse after drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?
(0) Never
(1) Less than monthly
(2) Monthly
(3) Weekly
(4) Daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
(0) No
(2) Yes, but not in the last year
(4) Yes, during the last year

10. Has a relative or friend or a doctor or another health worker been concerned about your drinking or suggested you cut down?
(0) No
(2) Yes, but not in the last year
(4) Yes, during the last year

Record total of specific items here

The Alcohol use disorders identification test (AUDIT)

The AUDIT has been established to provide an accurate degree of risk across various culture groups, gender and age. It was developed and evaluated over a period of 20 years due to its cross national standardization, the AUDIT is the only screening test specifically developed for worldwide use.

WHO developed AUDIT as a simple measure for screening of excessive alcohol consumption and to help in brief assessment in the wide range of health care settings. It can be used by both health care personals and non health care personals.

AUDIT was developed to identify excessive alcohol consumption pattern and to identify who are the people benefit from either reduction in alcohol consumption or cessation.

The AUDIT will identify three different alcohol related problems:

- ❖ Hazardous drinking
- ❖ Harmful drinking
- ❖ Alcohol dependence
- ❖ Hazardous drinking

There is no current medical problem in this group of patients. This type of drinking pattern is of public health importance. This pattern of alcohol drinking elevates the risk of harmful consequences for the drinker or others.

Harmful use.

Pattern of alcohol consumption that results in physical and mental harm to the user.

Alcohol dependence

It is usually develops after repeated alcohol consumption and is characterised by cluster of behavioural and cognitive and physiological phenomenon.

- ❖ Strong wish to drink alcohol
- ❖ difficult to control over its use,
- ❖ Continuous drinking in spite of harmful consequences.
- ❖ Top priority given to alcohol consumption than any other activity.
- ❖ Higher amount alcohol needed for desire effect
- ❖ Development of withdrawal reactions, once alcohol use is discontinued.
- ❖ These are clinical features of alcohol dependence.

Benefits of screening

It provides an opportunity to gather information about patients current condition and to evaluate the patients about risk of excessive alcohol consumption. It gives the opportunity to take preventive measures to reduce the alcohol related risks.

It has the following advantages over other tests:

1. It identify three component of alcohol use, harzadous alcohol use, harmful and also possible alcohol dependence.
2. It is brief rapid and flexible according to the situation.
3. It also focus on recent alcohol consumption

Domains and Item Content of the AUDIT		
Domains	**Question Number**	**Item Content**
Hazardous Alcohol Use	1	Frequency of drinking
	2	Typical quantity
	3	Frequency of heavy drinking
Dependence Symptoms	4	Impaired control over drinking
	5	Increased salience of drinking
	6	Morning drinking
Harmful Alcohol Use	7	Guilt after drinking
	8	Blackouts
	9	Alcohol-related injuries
	10	Others concerned about drinking

The audit scoring and interpretation

There are total of 10 questions, each question have the score of 0 to 4. Maximum score possible is 40. Dependent upon score, alcohol related problems are classified and managed accordingly.

In most studies, cut off of 8 provides very good sensitivity and acceptable specificity for identifying alcoholic disorder. The AUDIT is best screening tools in primary care settings in comparison with CAGE and MAST.

Advantages of Different Approaches to AUDIT Administration

Questionnaire	Interview
Takes less time	Allows clarification of ambiguous answers
Easy to administer	Can be administered to patients with poor
	reading skills
Suitable for computer administration	
and scoring	
May produce more accurate answers	Allows seamless feedback to patient
	and initiation of brief advice

Review of studies

John B Saunders et al (1993): When the AUDIT score of 8 or more used to diagnose AUD, Audit has sensitivity of 92% and specificity of 92% to diagnose hazardous or harmful alcohol use. Studied done in 1088 patients who were attending primary health care facilities.

Bohn Mu et al[40] (1995) done in a case control study shows that AUDIT score was superior to MAST to discriminating hazardous drinkers from non hazardous drinkers.

Gache P et al (2005)[41] study done simultaneously in France and Switzerland involving 1207 patients coming to the OPD. AUDIT shows better results than MAST and CAGE to diagnose alcohol abuse / dependence.

Personnel, Settings and Groups Considered Appropriate for a Screening Programme Using the AUDIT

Setting	Target Group	Screening Personnel
Primary care clinic	Medical patients	Nurse, social worker
Emergency room	Accident victims, Intoxicated patients, trauma victims	Physician, nurse, or staff
Physician's Room Surgery	Medical patients	General practitioner, family physician or staff
General Hospital wards Out-patient clinic	Patients with hypertension, heart disease, gatrointestinal or neurological disorders	Internist, staff
Psychiatric hospital	Psychiatric patients, particularly those who are suicidal	Psychiatrist, staff
Court, jail, prison	DWI offenders violent criminals	Officers, Counsellors
Other health-related facilities	Persons demonstrating impaired social or occupational functioning (e.g. marital discord, child neglect, etc.)	Health and human service workers
Military Services	Enlisted men and officers	Medics
Work place Employee assistance Programme	Workers, especially those having problems with productivity, absenteeism or accidents	Employee assistance staff

Biomarkers

Biomarkers have clinical usefulness for finding, diagnosis, and management of alcohol use disorders and also useful in screening for fetal alcohol exposure.

There are direct and indirect bio markers but most commonly used once are indirect bio markers.

Indirect biomarkers are:

Those that reflect the lethal effects of alcohol on organs, tissues, or blood biochemistry.

Indirect biomarkers include

- hepatic enzymes
- aspartate aminotransferase
- alanine aminotransferase
- gamma glutamyltransferase
- carbohydrate-deficient transferrin
- mean corpuscular volume.

Direct biomarkers are products of alcohol metabolism.

Direct biomarkers include

- acetaldehyde adducts
- ethyl glucuronide
- ethyl sulfate
- phosphatidylethanol
- fatty acid ethyl ester

TABLE 2. Generalized ranges of common lab tests for alcohol dependence.

TEST	SENSITIVITY (%)	SPECIFICITY (%)
CDT	60–70	80–95
GGT	40–60	80–90
MCV	30–75	60–90
AST	20–80	50–95
EtG	70–90	80–95
CDT + GGT	60–90	80–95
CDT + MCV	60–95	80–95

NOTE: Values obtained as a conglomerate of accepted values, review articles, and primary sources, including those listed in Table 1.

KEY: carbohydrate-deficient transferrin=CDT; gamma-glutamyl transferase=GGT; mean corpuscular volume=MCV; aspartate aminotransferase=AST; ethyl glucuronide=EtG

ASPARTATE AMINO TRANSFERASE (AST)

In old literature it was called as serum glutamate oxaloacetate transaminase (SGOT).AST needs pyridoxal phosphate (vitamin B6)as co-enzyme. Normal serum level of AST ranges from 8 to20 U/L. It is a marker of liver injury and shows moderate to drastic increase in parenchymal liver diseases like hepatitis and malignancies of liver.

AST was used as a marker of myocardial ischemia in olden days. The level is significantly elevated in myocardial infarction. But troponins have replaced AST as a diagnostic marker in ischemic heart disease

ALANINE AMINO TRANSFERASE (ALT)

In old literature, it was called as serum glutamate pyruvate transaminase (SGPT). The enzyme needs pyridoxal phosphate as coenzyme.

Normal serum level of ALT for male is 13-35 U/L and for female is 10-30 U/L. Very high values (300 to 1000 U/L) are seen in **acute hepatitis,** either toxic or viral in origin.

Both ALT and AST levels are increased in liver disease, but ALT > AST. Rise in ALT levels may be noticed several days before clinical signs such as jaundice are manifested. Moderate increase (50 to 100 U/L) of ALT may be seen in chronic liver diseases such as cirrhosis, hepatitis C and non-alcoholic steatohepatitis (NASH).

Normal AST: ALT ratio is 0.8. A ratio >2 is seen in
Alcoholic hepatitis
Hepatitis with cirrhosis
Nonalcoholic steatohepatitis (NASH)
Liver metastases
Myocardial infarction
Erythromycin treatment
A low ratio (ALT is higher) is seen in
Acute hepatocellular injury
Toxic exposure
Extra hepatic obstruction (cholestasis

Gamma Glutamyl Transferase (GGT)

GGT is clinically important because of its sensitivity to detect **excess alcohol consumption**. GGT level in alcoholic liver disease roughly parallels the alcohol intake Elevated levels of GGT are observed in chronic alcoholism, pancreatic

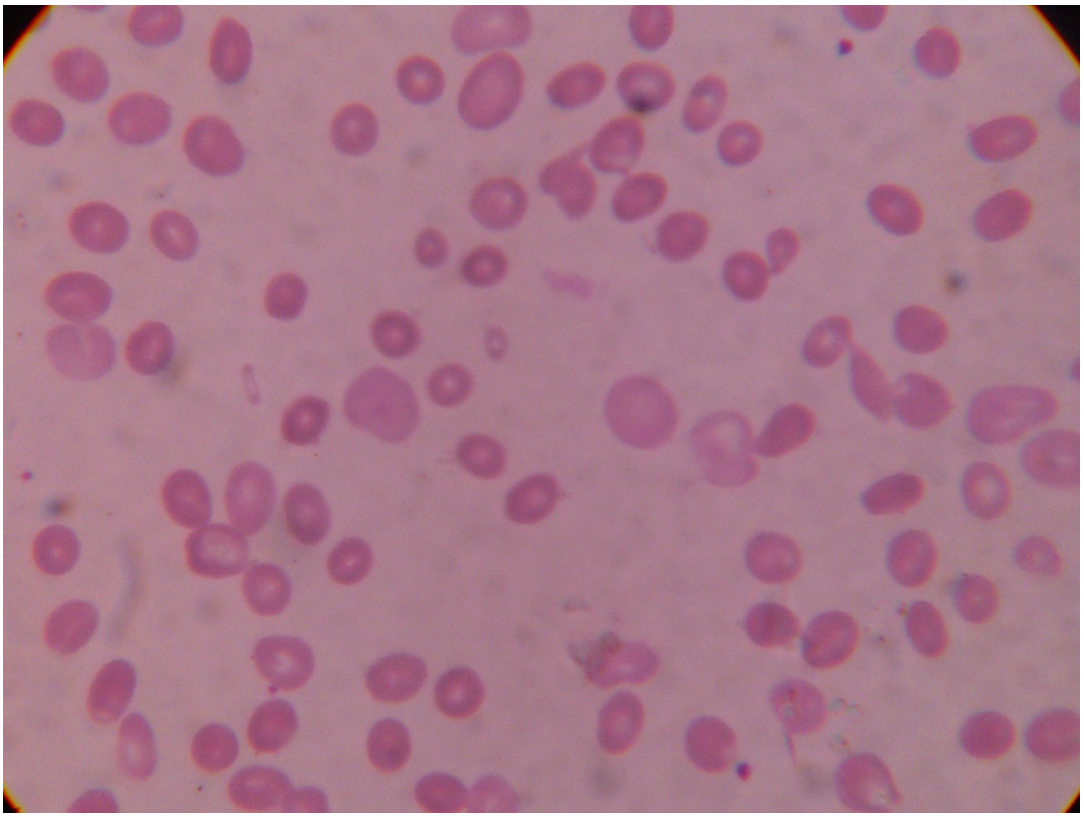
disease, myocardial infarction, renal failure, chronic obstructive pulmonary disease and in diabetes mellitus.[27,28] In liver diseases, GGT elevation parallels that of ALP and is very sensitive of biliary tract disease. The old name was gamma glutamyl transpeptidase. It can transfer gamma glutamyl residues to substrate. In the body it is used in the synthesis of glutathione. GGT has 11 iso-enzymes

Normal serum value of GGT is 10-30 U/L. It is moderately increased in infective hepatitis and prostate cancers. In alcoholics even when other liver function tests are within normal limits. GGT level is rapidly decreased within a few days when the person stops to take alcohol. Increase in GGT level is generally proportional to the amount of alcohol.

Other liver function tests like serum bilirubin , alkaline phosphtsase , albumin , Prothrombin time not useful as a bio marker unless complicated by liver disease. Once liver disease established these markers may be useful to categorize decompensated liver disease

Mean Corpuscular Volume[43,44,45]

The presence of mean corpuscular volume may be an indicator of underlying disease. An elevated MCV is referred as macrocytosis upper reference limit for mean corpuscular volume varies from 95 to 100 fl. It depends upon age and laboratory. Generally laboratories often gives upper reference limit as 100 fl[43].



Cause of Macrocytosis

Vitamin B12 and folate deficiency
Alcohol
Liver disease
Bone marrow disorders (myelodysplasia, aplastic, dyserythropoietic and sideroblastic anaemias and leukaemia)
Medications
Physiological (neonates, pregnancy)
Hypothyroidism
Artifact (cold agglutinins, hyperglycaemia)
Hyperlipidaemia
Reticulocytosis
Postrenal transplant

CAUSE	PERCENT OF PATIENTS BY STUDY				
	DAVIDSON * (N=200)	BREEDVELD * (N=70)	KEENAN * (N=80)	SAVAGE * (N=300)	MAHMOUD * (N=124)
Alcohol	18	27	36	26	14
Vitamin deficiency	13	39 (6% had both deficiencies)	16	6	24
B_{12}	8	23	10	5	12
Folate	5	10	6	1	12
Medications	30	1	—*	37	2
Liver disease	16	3	9	6	2
Hematologic disease	15	19	14	14	20
Malignancy/premalignancy	15	13	11	6	20
Reticulocytosis	0	6	3	8	—†
Hypothyroidism	—†	3	6	1	12
Unexplained	23	9	28	7	19
* Excluded patients on cytotoxic and chemotherapeutic medications					
† Not evaluated.					

Mean corpuscular volume (MCV) has also long been known to elevate with chronic excessive alcohol intake. This elevation appears to be mainly because of direct morrow-toxic effects of alcohol, alcohol does affect folate absorption, a true folate deficiency occurs in only 17 percent of chronic alcoholics, while elevated MCV is more common. And with an red

blood cells half life 120 days, elevated MCV is a marker of more chronic alcohol ingestion and it is not a good marker of acute ethanol ingestion or acute relapse[13].

Sensitivity is between 30-75% in varies studies. Elevated MCV has also been shown to be more sensitive than men in women for unknown reasons.. An elevated MCV above the normal range has a very good specificity, often quoted as over 90 percent.[13,15]. Since MCV is another readily available, investigation its use should encouraged when a patient came with a features of chronic alcohol abuse and dependence[13]. Interestingly, in combination with AST, a normal lab value virtually rules out delerium tremens in emergency patients[14].

Review of studies

Seppä K.et al 1991[16]macrocytosis may be the only indicator of chronic alcohol ingestion , particularly in young and middle-aged men.

Mundle G et al 2000[17] the "forgotten" marker MCV is superior in women to find out alcohol use disorder and it is a marker of second choice in men. The combination GGT and MCV is the most cost-effective choice for men and women.

Kumar BG Prashanth et al2012[18] The study shows that elevated MCV to be possessing **sensitivity87.5% andspecificity of, 83.33%**. positive predictive value is 87.5% and negative predictive value 48.39%. Diagnostic accuracy of the test to find out alcohol use disorder 54.29%

They are also helpful in long term follow-up of patients undergoing treatment, and monitoring of abstinence beyond 120 days

carbohydrate-deficient transferrin (CDT)

Transferrin is a protein synthesized by the liver. It involved in iron transportCDT refers to isoforms that are deficient in sialic acid. CDT is defined as the monosialo, disialo, andasialo isoforms of transferrin. Excess alcohol consumption increases the fraction of CDTs[19,20].

CDT test has moderate sensitivity and specificity and is a longer-term marker of excess alcohol consumption ingestion of 50 to 80 g alcohol per day for 1 to 2 weeks elevates CDT abovebaseline[21]because of its relative short half life CDT is especially useful as a marker for abstinence . and came back to baseline levels 2 to 5 weeks after alcoholcessation.

The most important factor influencing CDT testing accuracy to detect alcohol use disorder is gender. more false positives test results can occurs in women even with the newer % CDTtests[24,25].

Review of studies

Sorvajärvi K. **etal** 1996[22] alteration in serum total transferrin levels affect the specificity CDT assay. This should be kept in mind when interpreting the CDT results in patients with elevated serum transferrin, such as iron deficiency anaemia, pregnancy, and liver diseases.

Radosavljevic M et al 1995[23] an elevated carbohydrate deficient transferrin level, however, cannot be taken as reliable confirmation for chronic alcohol abuse in patients with hepatic disease.

GURUPRASAD P et al1998[26]in patients with a "**wide range of alcohol intakes"** bio markers such as serum GGT and MCV were more appropriate than serum CDT for measuring alcohol intake. Serum CDT when used in combination with serum GGT and MCV was useful in finding out excessive alcohol intake. The significance of careful alcohol history with a standardized questionnaire is highlighted.

NEED FOR THE STUDY

Physicians usually identify only half of patients with drinking problems for the reason that of both insufficient time spent on questioning by doctors and denial by patients[9]. Underestimation of alcohol related problem is particularly common in older patients[10].

Measuring alcohol consumption in an individual has so many restrictions. Most studies depend on interviews with patients and their family members to assessment the amount, frequency and extent of alcohol intake.

Patients may not accurately report the amount of alcohol they consume[8] and the definition of a 'standard drink' varies from region to region[11].

Patients with compensated liver disease generally do not have any symptoms so they do not seek medical attention. Alcohol-related problems are usually not detected until late when the developed decompensated liver disease[12]. “PREVENTION BETTER THAN CURE. BY EARLY DETECTION WE CAN PREVENT AHE ALCOHOL DEPENDENCE AND ALCOHOL RELATED OTHER PROBLEM” symptoms so they do not seek medical attention. Alcohol-related problems are usually not detected until late when the developed decompensated liver disease[12].

MATERIALS AND METHODS

This is a cross sectional study and it was conducted at the Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for a period of six months from June 2013 to November 2013. The protocol for this study was approved by the Institutional Ethical Committee (IEC), Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3.

INCLUSION CRITERIA

For participation in this study:

Patients admitted in the medical wards found to having alcohol use disorders which was identified by using the AUDIT score.

EXCLUSION CRITERIA

- Patients not willing to participate in the study after clear explanation.
- Patients who are having anemia.
- Patients who are having thyroid disorders.
- Pregnant women
- Patients who are taking anti epileptic drugs.
- Patients who are taking chemotherapeutic drugs.
- Patients who are antiretroviral drugs

After meticulous assessment of inclusion and exclusion criteria, 100 patients were enrolled in this study.

Verbal explanation about this study was clearly enlightened to the participants and the doubts in this study was heard and clarified. From those who are willing to enrol in this study an informed consent in the prescribed format which was submitted to the ethical committee was obtained.

Participants who cannot understand the English language, a consent was obtained in the mother tongue (Tamil), so that the participants can have better understanding about the study details.

From the eligible subjects, history related to current medical problem, past history, personal history pertaining to smoking, drug abuse and high risk sexual behaviour were recorded. Occupational history was also obtained.

Alcoholic History

Detailed history of alcohol intake was recorded as per AUDIT proforma interview version by World Health Organization. Age of initiation of alcohol and family history of alcohol use disorders were also obtained. It was explained about what is standard drink and questions are asked as per proforma. Total of 10 questions asked and score given 0 to 4 according to their response. Sub scores were made according to the questions answered.

Consumption score was made by adding up of question numbers 1 to 3, total sub score possible is 12. Any score more than 6 is significant. Dependence score was made by adding up to scores of answer given to

question numbers 4 to 6 and total sub score possible is 12. Score more than 4 is significant.

Alcohol related problem score was made by answer given to question numbers 7 to 10 and total score possible is 16.

Risk stratification which is useful in management done as per score given in the below tables:

Risk Level	Intervention	AUDIT score
Zone I	Alcohol Education	0-7
Zone II	Simple Advice	8-15
Zone III	Simple Advice plus Brief Counseling and Continued Monitoring	16-19
Zone IV	Referral to Specialist for Diagnostic Evaluation and Treatment	20-40

These subjects were subjected to general examination, measurement of vitals and systemic examination to find out alcohol related problems and any other associated medical problem. Measurement of blood pressure was done in a quiet room patient was seated position for five minutes, well calibrated and accepted blood pressure machine with appropriate cuff size, working on mercury scale was used to measure the resting blood pressure. Blood pressure was recorded in the two arms and the highest of these two recording were mentioned. Patient who were on medication for blood pressure control were allowed to take morning dose of scheduled drugs and blood pressure was recorded under protective cover of anti hypertensive only.

Assays and Calculations:

Under strict aspetic precaution, sufficient blood samples were drawn, biochemical analysis which includes random blood sugar, blood urea, serum creatinine and liver function test which includes serum bilirubin, aspartate amino transferase, alanine amino transferase, alkaline phosphoatase, serum albumin were done by auto analyser at central biochemistry laboratory at Rajiv Gandhi Government General Hospital, Chennai. For complete blood count, blood samples were collected from ante cubital vein using syringe and transferred to EDTA tube and analysed in automated cell counter sysmex KX21 and anslysed.

Peripheral smear was done at Pathology Lab at Rajiv Gandhi Government General Hospital, Chennai-3. Ultrasonagram of abdomen was done at Bernard Institute of Radiology, Rajiv Gandhi Government General Hospital, Chennai-3.

Criterias used were:

Blood Pressure $\geq$140/90 mmHg or <140/90 mmHg with anti hypertensive medications as per JNC VII guidelines (Joint National Committee on prevention, detection, evaluation and treatment of High Blood Pressure).

Total AUDIT score 8 and above is taken as alcohol use disorders.

Mean Carpuscular volume of >100 fl as taken as macrocytosis.

Statistical methods

Data reported in this study includes descriptive statistics of participants at the time of enrolment. Qualitative variables are analysed by chi-square test. The quantitative data in this study were analysed using independent sample t test.

Sponsorship

Nil

Conflict of Interest

None

OBSERVATIONS AND RESULTS

Table 1 : AGE GROUP AND MACROCYTOSIS

Age group (in yrs)	MCV >100fl	MCV <100fl
20 – 30	07	06
31 – 40	20	14
41 – 50	20	12
51 – 60	08	07
>60	04	02

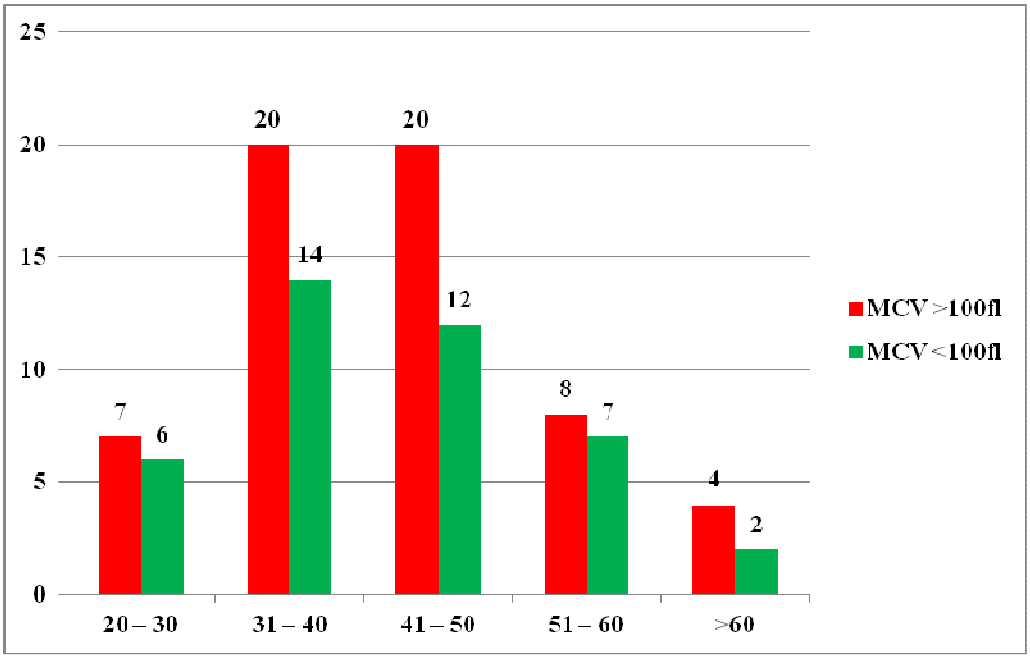


Table 2 : ALCOHOL USE DISORDER AND MACROCYTOSIS

Alcohol use disorder (No. of patients)	MCV >100fl	MCV <100fl
100	61	39

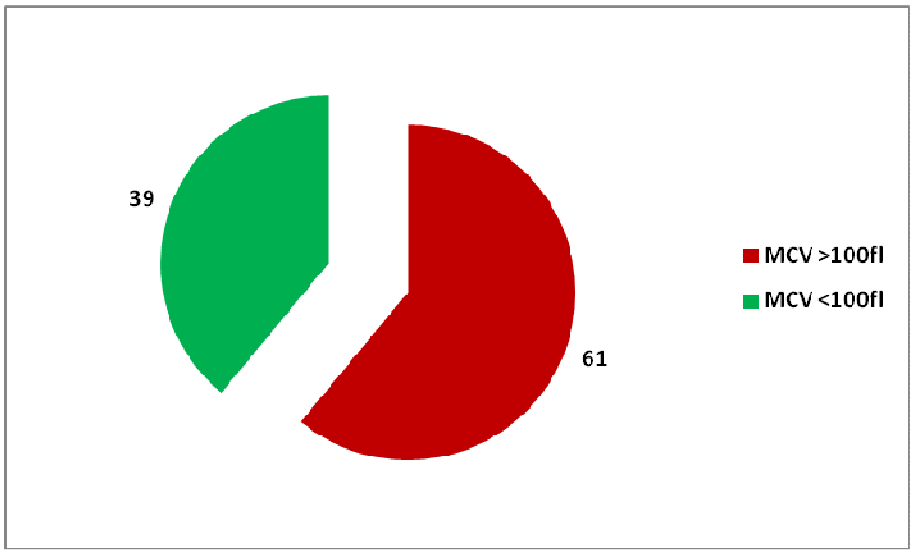


Table 3 : RISK STATIFICATION USING AUDIT SCORE AND MACROCYTOSIS

Zone	MCV >100fl	MCV <100fl
Zone I (Score 0 – 7)	0	0
Zone II (Score 8 – 15)	04	06
Zone III (Score 16 – 19)	07	05
Zone IV (Score 20 – 40)	50	28

RISK STATIFICATION USING AUDIT SCORE AND MACROCYTOSIS

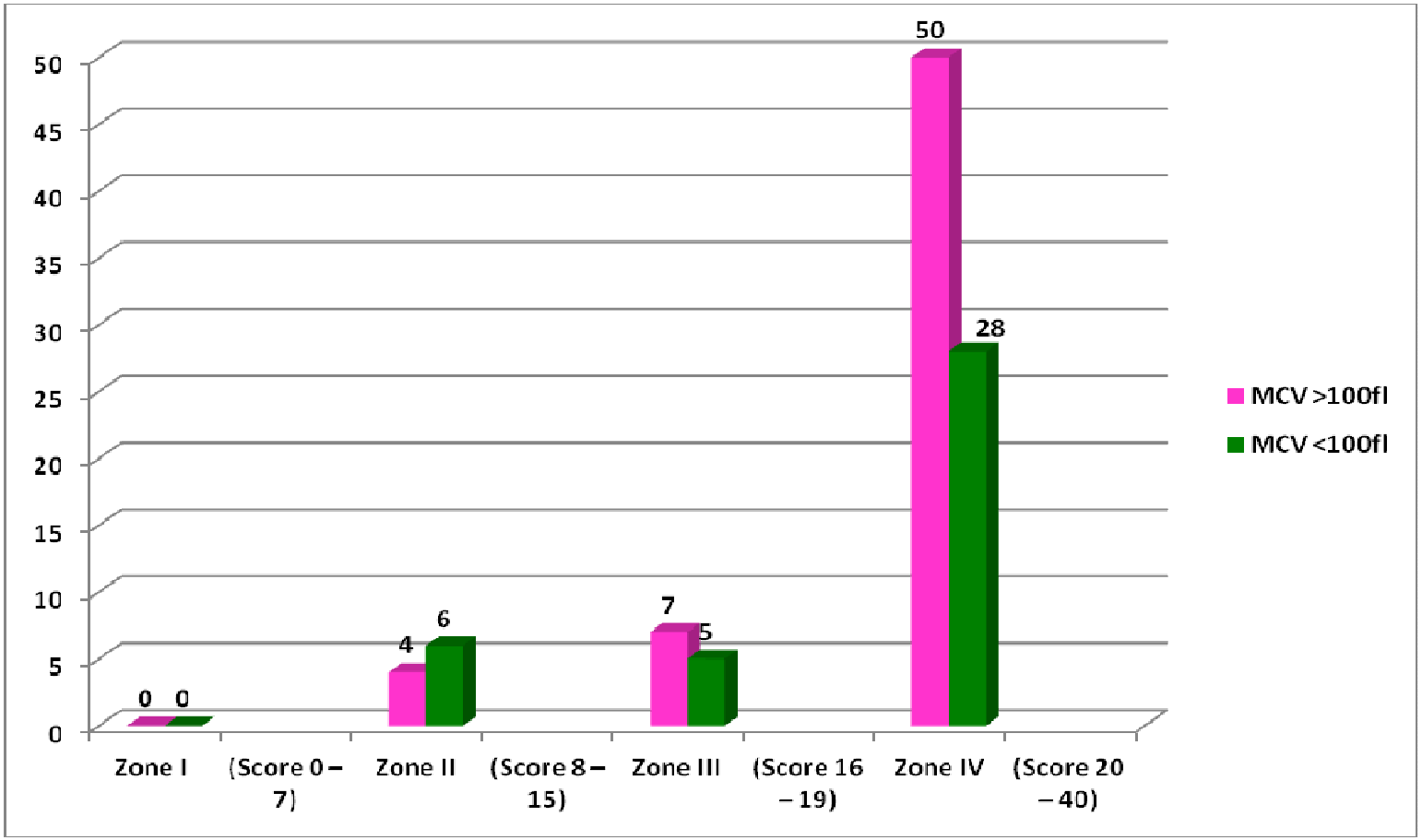


Table 4 : AST / ALT RATIO AND MACROCYTOSIS

	AST/ALT ratio <1	AST/ALT ratio 1 – 2	AST/ALT ratio >2
MCV <100 fl	09	25	05
MCV >100 fl	10	37	14
Total	19	62	19

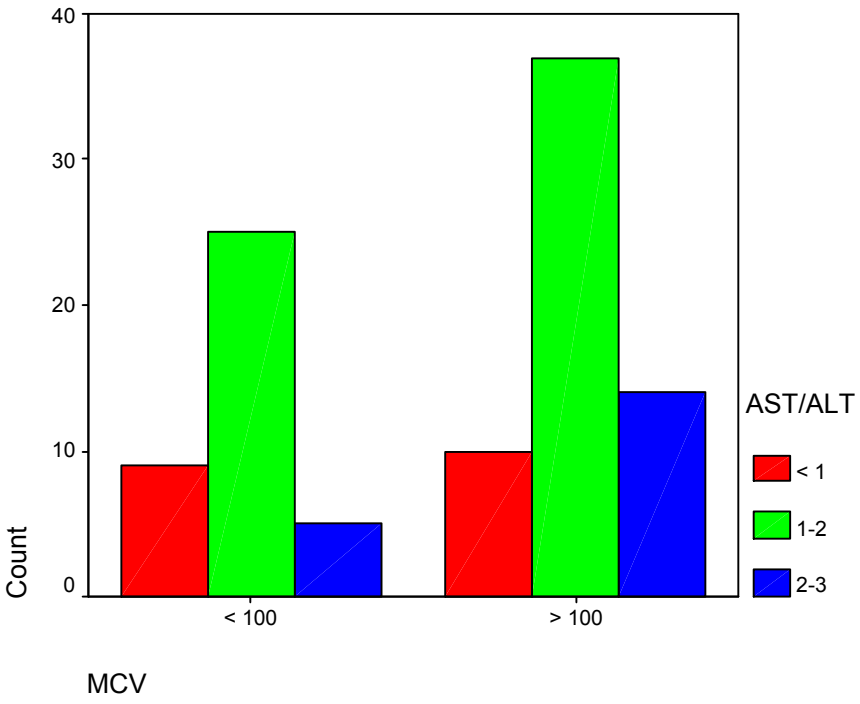


Table 5 : Alcohol use disorders in medical ward patients and admission diagnosis

Sl.No.	Diagnosis	No.of patients (%)
1.	Hepatobilliary	36%
2.	Neuro-psychiatry	32%
3.	Infection	17%
4.	Gasterointestinal	15%
5.	Cardiovascular	7%
6.	Self harm	3%

Alcohol use disorders in medical ward patients and admission diagnosis

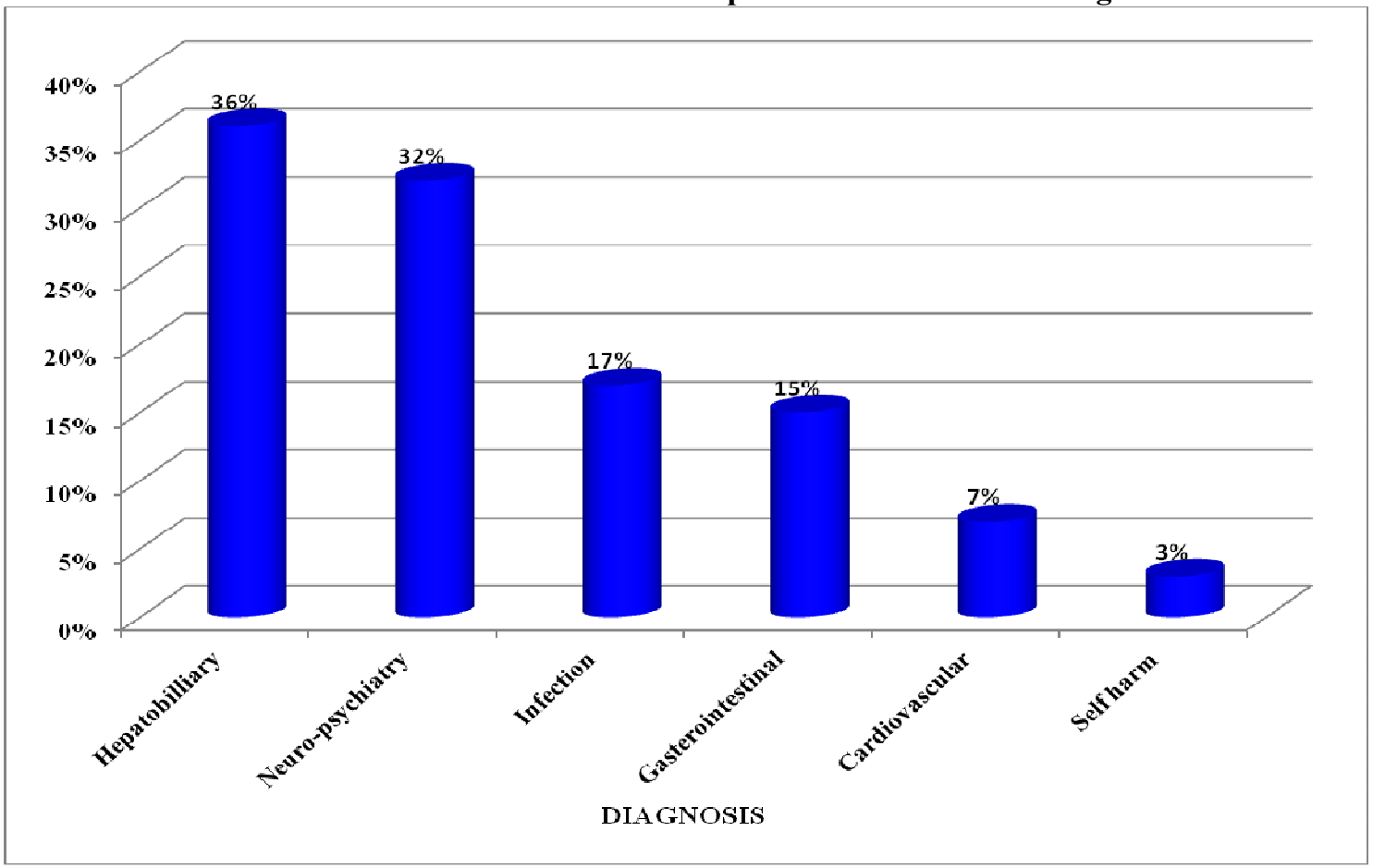


Table 6 : ALCOHOL USE DISORDER AND HYPERTENSION

Alcohol use disorder (No. of patients)	Systolic Hypertension	Diastolic Hypertension
100	25	33

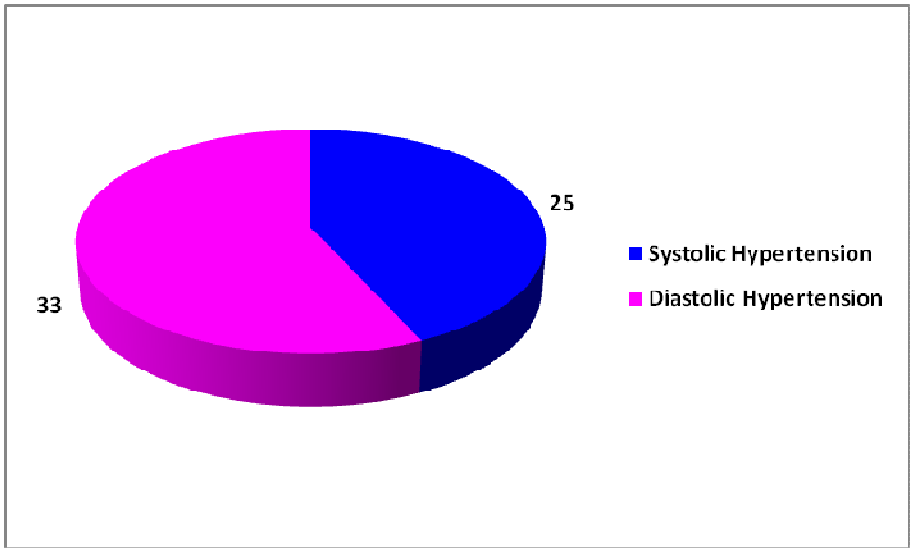


Table 7 : ALCOHOL USE DISORDERS AND OCCUPATION

Sl.No.	Occupation	No.of patients (%)
1.	Others	43%
2.	Drivers	20%
3.	Construction workers	17%
4.	Hospitality	8%
5.	Other daily wagers	7%
6.	Students	4%
7.	Unemployed	1%

ALCOHOL USE DISORDERS AND OCCUPATION

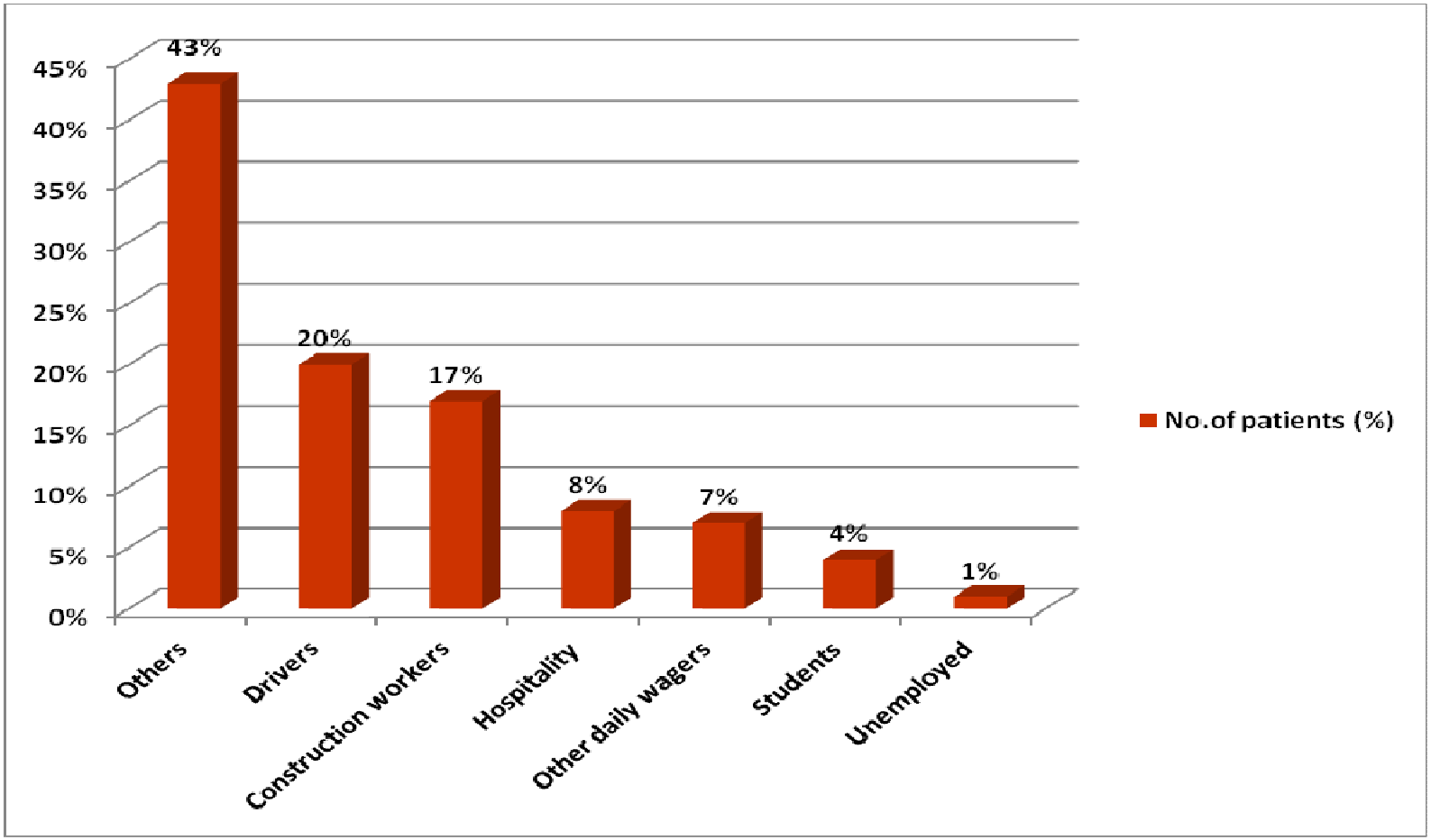


Table 8 : ALCOHOL USE DISORDERS AND USG ABDOMEN FINDIDNGS

Sl.No.	USG abdomen findings	No.of patients (%)
1.	Fatty Liver	41%
2.	Normal Study	34%
3.	DCLD	23%
4.	Others	2%

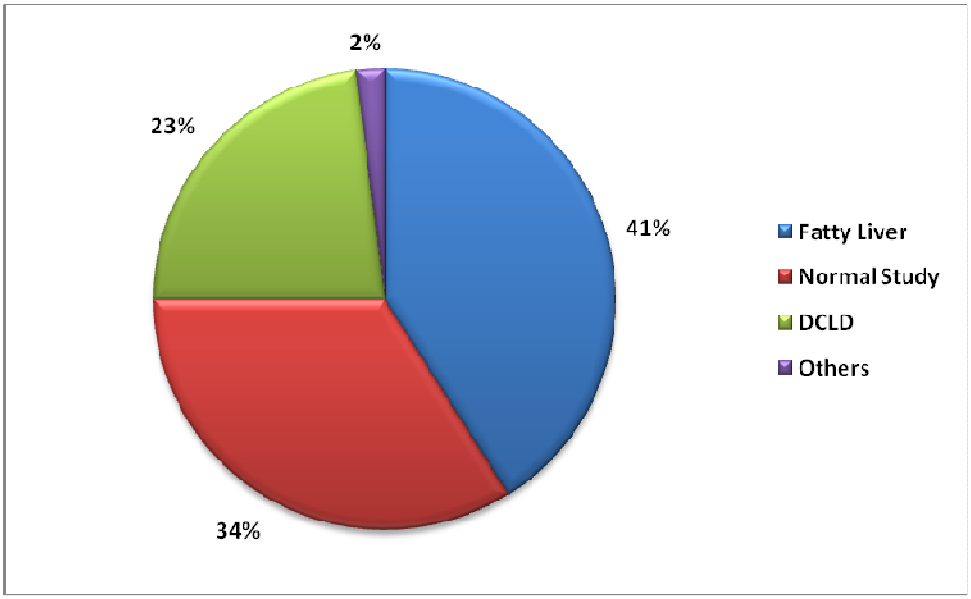


Table 9 : ALCOHOL USE DISORDERS AND SMOKING

Alcohol use disorder (No. of patients)	Smokers	Non Smokers
100	52%	48%

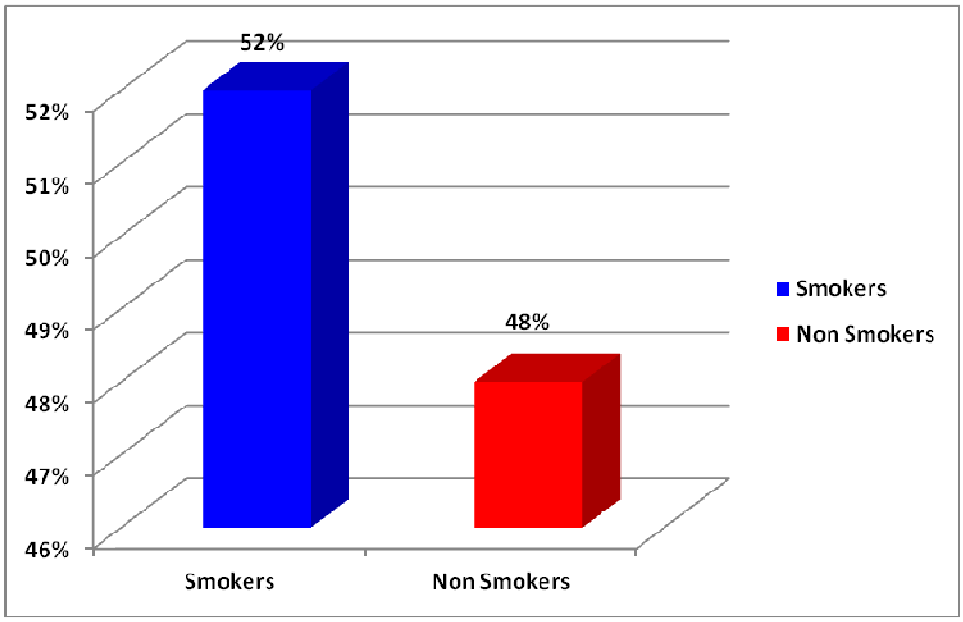
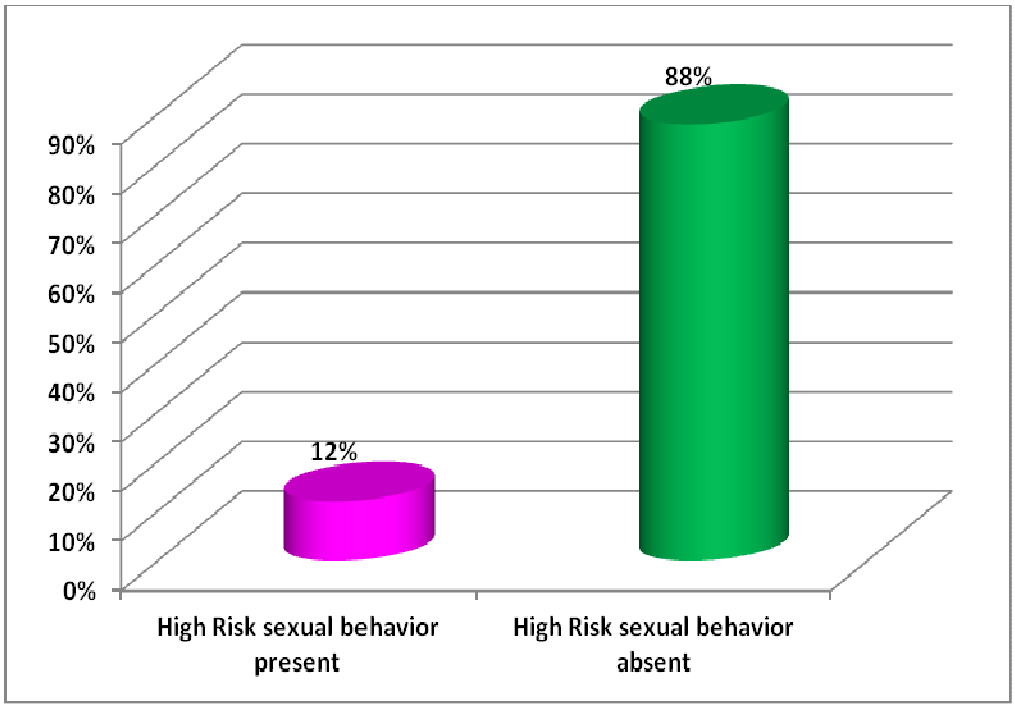


Table 10 : ALCOHOL USE DISORDERS AND HIGH RISK SEXUAL BEHAVIOUR

Alcohol use disorder (No. of patients)	High Risk sexual behavior present	High Risk sexual behavior absent
100	12%	88%



RESULTS

Total number of participants in this study was 100 in number who were found to having alcoholic disorders. An analysis of these group of people, macrocytosis was noted in 61% and the remaining 39% had normal mean corpuscular volume.

Age and Macrocytosi

In this study, alcoholic disorder prevalence is higher among age group of 31 to 40 years (34%) and 41 to 50 (32%) contributing around 66% of patients. Alcohol use disorders is prevalent in the productive age group but there is no statistical difference among age grouping macrocytosis. The youngest one noted in this study was 20 years and elder one was 75 years. So, problem is common in all age groups.

Risk stratification using AUDIT score and macrocytosis

Risk stratification done by total AUDIT score shows that 78% of patients were in high risk zone who requires immediate intervention among patients belong to high risk zone 50 had macrocytosis and 28 had normal mean corpuscular volume. 12 patients were belong to the moderate risk group who requires counselling for alcohol related problems. 7 of them had macrocytosis, 5 had normal mean corpuscular volume. 10 patients belong to zone II. These patients needs simple advise for alcohol related problems. Patients having more AUDIT score showing statistically significant p value (0.028).

AST / ALT ratio and macrocytosis

AST/ALT is one of the biomarker to identify alcohol related problems. AST/ALT ratio between 1 to 2 presented in 62% of people, among them macrocytosis presented in 60.7%. AST/ALT ratio of more than 2 presented in19 patients, among them 14 patients had macrocytosis which contribute to 73.7% patients in that group.

Using chi-square test, AST/ALT ratio 1 to 2 have significant associated with macrocytosis with p value of 0.0001.

Alcohol use disorder in medical ward inpatient

Patients who were admitted in medical wards who were found to having alcohol use disorders which was confirmed by AUDIT shows that 36% of patients had hepatobillary system involvement. In the hepatobillary system decompensated liver disease contributes to 23 cases and 1 patient had hepatocellular carcinoma.

32% of patients admitted had neuro psychiatric problems like alcohol intoxication, alcohol withdrawal and others.

Next major contributor is infection. It causes 17% of patients. Gasterointerstinal system involved in 15% of patients. It is mainly due to alcoholic gastritis. One had esophageal carcinoma and one had carcinoma stomach.

Alcohol known to cause high risk behaviour, self harm presented in three patients. One had attempted hanging, two admitted for allerged consumption of poisoning.

Alcohol use disorder and hypertension

In the total of 100 patients, 33 patients had diastolic hypertension and 25 had systolic hypertension. 2 patients admitted for emergency management of accelerated hypertension. Among 33 patients, only 20 patients knows that they were having hypertension. In the 20 patients, only 15 patients were on the regular medications.

Alcohol use disorder and Occupation

Alcohol use disorders were widely prevalent in all socio economic groups. Previously it thought that it was prevalent only in low socio economic groups. It uniformly spread among all groups. 37% patients were contributed by drivers (20%) and construction workers (17%). Students contributed 4%. Other daily wagers like load man, agriculturist contributes to 7%. Persons who were working in hotels, bar are more prone to develop AUD at younger age group because of easy availability of alcohol. 8% of patients were belong to these groups. In this study one person is unemployed. The remaining 43% distributed evenly in all other categories.

Alcohol use disorder and USG abdomen findings

A well known fact that alcoholism leads on to fatty liver disease. It is prevalent upto 90%, but in our study 41% of patients had fatty liver, 23%

patients had decompensated liver disease which is the advanced spectrum of fatty liver disease. Normal study was noted in 34% of people, one person had bilateral medical renal disease.

Alcohol use disorder and smoking

In our study group, 52% of patients used tobacco mainly in the form of smoking and they continue to using regularly. 48% were non smokers. None of the person had any other oral or IV drug abuse history.

Alcohol use disorder and high risk sexual behaviours

In our study group, 12% patients had high risk sexual behaviour like multiple partners, unprotected sex and MSM (Man seeking Men) sex. Among this 12 patients, 3 patients diagnosed to having retroviral disease after admission. 88% of patients did not had high risk sexual behaviour.

DISCUSSION

In our study, the prevalence of macrocytosis in patients with AUD is 61% with p value of 0.028.

As per study done by GA Gomex et al[50], the study conducted in hospitalised inpatients both compressing of medical and surgical patients shows that mean corpuscular volume as a sensitivity and specificity of 74% to diagnose alcohol use.

Kumar PG Prashanth et al (2012) done a case control study in India involving 40 cases and 30 controls shows that elevated MCV has 87.5% sensitivity and specificity. MCV also useful in monitoring long term abstinence[51].

As per study done by S.Chaudhury et al, a case control study conducted in India, 100 male inpatients admitted in psychiatric hospital for alcohol dependence shows that MCV sensitivity of 31% and specificity of100%[52].

As per summary released by National Institute of Alcohol abuse and alcoholism shows that MCV has sensitivity of 47% to diagnose AUD. The study was done by Anttila et al 2004.

A study done by Seppa K et al in 1991, macrocytosis as a common findings in patients who were not anaemic and without other haematological abnormalities was generally ignored. The study involved 300 patients

attending general practitioners who has macrcytosis. 80.2% of men and 34.1% of women are alcohol abusers.

As per Seppa K et al (1992) comparison of alcohol use- macrocytosis and blood pressure case study involving 95 patients and 22 controls shows that 72% of men with macrocytosis were alcoholic and 41 of them had either systolic or diastolic blood pressure. In female patients, the systolic blood pressure is normal and slight elevation in diastolic blood pressure. In our study, prevalence of diastolic hypertension is 33% and systolic hypertension is 25%.

Pandey et al conduct a prevalence study with a help of Indian Council of Medial Research conducted in patients who had sexually transmitted disease. 26% of patients who were having sexually transmitted disease were alcoholic user. In our study, three persons had retroviral disease.

As per Nyblom H et al study done at three different groups, first group comprises of patient with alcoholic dependence. The total number of patients were 313. Second group has 78 patients with the history alcohol abuse and third group 48 patients with alcohol abuse and cirrhosis and its complications. The study shows that patient without liver disease does not have high AST/ALT ratio. AST/ALT ration above 1 indicator for advanced liver disease. In our study 81% of patients AST/ALT ratio more than 1. 62% of patients had ratio between 1 and 2 and 19% had ratio more than 2.

It may be because of high prevalence of hepatobillary system involvement in our study.

As per study Mohan D et al (2002) done in representative of general population in capital of India. Over 5414 men and 4898 women shows that prevalence of concurrent smoking and alcohol is 9.6% in general population.

LIMITATIONS OF STUDY

One limitation of our study involved low number of participants in comparison to other studies. Hence, these results need to be confirmed in large group of patients and longitudinal studies. There were no women participants in this study to compare the effect of AUD and macrocytosis in them.

CONCLUSION

Diagnosing alcohol injection after development of alcohol dependence or alcohol related problems has no value in the management. A combined use of AUDIT and cost effective investigations like mean corpuscular volume. We can diagnose the problem earlier and we can prevent the complications.

Patients with macrocytosis without anaemia should be evaluated for alcohol abuse.

Mean Corpuscular volume the only biomarker that reduce long time after alcohol cessation. It has to be studied to follow up of long term abstinence.

Large community based studies needed involving alcohol users without complications to find out usefulness of this test.

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ABBREVIATIONS

AUDIT	**:**	**Alcohol Use Disorders Identification Test**
MCV	**:**	**Mean Corpuscular Volume**
AUD	**:**	**Alcohol Use Disorders**
AST	**:**	**Aspartate Amino Transferase**
ALT	**:**	**Alanine Amino Transferase**
gm	**:**	**Grams**
dL	**:**	**Deciliter**
IU	**:**	**International Unit**

PROFORMA

Name	:	Age :	Sex:
IP No.	:	Occupation:	
History of present illness	:		
Past History	:		
Personal History	:		
Alcohol History as per AUDIT	:		
General Examination	:		
Systemic Examination	:		
Investigations	:		
CBC	:		
Peripheral Smear	:		
RFT	:		
LFT	:		
Thyroid Function Test	:		
USG abdomen	:		

MASTER CHART

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
1	Manikanadan	27	M	65196	H	-	-	130	90	19	7	8	4	87.3	38	40	<1	1.2	12	3.5	NSY	AG	H
2	Arumugam	26	M	66459	C	-	-	100	70	26	9	8	9	106.5	329	109	>2	23.8	109	4.6	Fatty liver	AI	NS
3	Venkatesan	32	M	68827	D	-	-	100	70	25	10	6	9	128.6	302	118	>2	2.2	160	4.5	Gr.I Fatty liver	ChP	Hepato billary
4	Vinoth	26	M	68903	S	-	-	110	70	10	6	4	-	113.1	60	48	>1	1.3	72	4	GB Wall thickening	Fever	infection
5	Prakash	35	M	68903	H	+	-	120	90	32	12	6	12	102.6	146	74	>1	1.6	64	3.7	Fatty liver	AI	NS
6	Suresh	32	M	63951	D	+	-	140	90	26	10	6	19	88.2	37	33	>1	0.9	95	3.9	NSY	AG	GIT
7	Venkatesan	40	M	64281	H	+	-	100	70	24	10	6	8	87.6	86	31	>2	1.3	30	3.8	Liver absess	AL	Hepato billary
8	Veeraragavan	42	M	64266	C	+	-	90	70	26	12	6	8	102	42	38	>1	1.2	31	3.2	NSY	SVT	CVS
9	Seenu	40	M	68909	U	-	-	130	80	22	10	6	6	81.4	116	100	>1	13.7	300	3.6	NSY	AWS	NS
10	Arunachalam	55	M	68956	O	-	-	140	90	22	10	6	6	102.2	42	40	>1	1.2	72	3.4	fatty liver	AWS	NS
11	Venkatesan	52	M	70641	C	-	-	100	70	18	8	4	6	86.5	97	88	>1	1.4	97	2.5	NSY	TB	Infection
12	Devadoss	35	M	113051	D	+	-	140	100	20	8	6	6	101.5	239	345	<1	20.8	525	3.4	Hepato cellular carcinoma	HCC	Hepato billary

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
13	Jayasingh	43	M	77265	C	+	-	100	60	27	10	7	10	97.5	92	46	>2	5.2	157	2.6	DCLD	DCLD	Hepato billary
14	Arumainathan	40	M	78666	O	-	-	100	70	30	10	10	10	92.6	51	41	>1	4.1	90	3.7	DCLD	DCLD	Hepato billary
15	Vardharajan	40	M	78830	O	+	+	90	60	20	8	6	6	78.7	34	35	<1	1.2	130	3	NSY	Alcoholic DCM	CVS
16	Kasi	46	M		O	-	-	140	90	26	12	8	6	104.6	48	42	>1	1.7	124	3.8	Fatty liver	ALD	Hepato billary
17	Sridhar	65	M	73700	O	+	-	150	100	30	10	6	16	106.1	78	46	>1	3.2	46	2.8	Fatty liver	AWS	NS
18	Yuvaraj	33	M	71297	O	-	-	140	90	32	10	8	14	90.9	178	187	<1	4.2	187	3.8	Fatty liver	AWS	NS
19	Mathesh	45	M	71232	O	+	-	130	80	28	10	8	10	116.8	42	36	>1	1.2	52	3	NSY	AG	GIT
20	Gunasekar	44	M	71327	O	-	-	100	70	28	10	6	12	103.3	69	270	<1	10	286	3.2	DCLD	DCLD	Hepato billary
21	Sakthivel	39	M	72431	O	-	-	130	80	30	10	8	12	95.4	59	46	>1	0.9	101	3.9	Fatty liver	AG	GIT
22	Sekar	46	M	81204	O	-	-	110	70	32	12	8	12	87.5	402	196	>2	6.8	206	2.5	Fatty liver	AH	Hepato billary
23	Raghu	42	M	81264	O	-	+	100	60	20	10	4	6	106.2	32	26	>1	0.8	134	3	NSY	Lung Absess	Infection

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	
24	Marriappan	38	M	81365	D	+	+	100	70	26	10	6	10	97.4	42	40	>1	8.6	48	2.2	DCLD	DCLD	H b
25	Kuppan	55	M	83546	O	+	-	94	60	26	10	6	10	112.2	40	32	>1	2.8	65	3.7	DCLD	DCLD	H b
26	Nehru	56	M	83802	O	-	-	100	70	28	10	8	10	107.6	88	42	>2	4.8	106	3	DCLD	DCLD	H b
27	Venkatesan	37	M	83906	C	-	-	130	100	24	10	8	6	110.6	42	36	>1	1.9	60	3.5	NSY	AG	
28	Ramesh	28	M	83957	D	+	+	140	90	32	12	8	12	93.7	92	86	>1	8.6	116	3.2	Fatty liver	AH	H b
29	Palanikumar	38	M	86061	H	-	-	100	70	25	9	6	10	116	46	36	>1	1.8	206	3.2	DCLD	DCLD	H b
30	Sivaprakash	39	M	86002	C	+	-	104	70	19	8	5	6	101.1	60	46	>1	2.7	99	4	DCLD	DCLD	H b
31	Ramu	52	M	88741	H	-	-	160	100	30	12	8	10	86.5	40	42	<1	0.9	26	3.2	NSY	AWS	
32	Kannan	48	M	86123	H	+	-	90	60	16	6	4	6	105.9	52	50	>1	1.3	67	2.9	ascites	CAD DCM DM	
33	David	40	M	88735	O	-	-	100	70	22	10	5	7	90.9	64	62	>1	1.6	106	3	Secondary Liver	Ca stomach Gr.IV	
34	Sudhakar	32	M	91321	D	-	-	114	70	20	8	6	6	92.3	38	40	<1	1.2	56	3	NSY	RVD	In

Sl.No.	Name		Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Audit Score			Investigations								Diagnosis	System
						Smoking	High risk sexual behaviour	SBP	DBP	Score (AUDIT)	Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	
35	Gowthaman	56	M	912456	O	+	-	150	100	35	12	10	13	104.8	88	43	>2	1	76	3.5	NSY	PN	
36	Ramesh	42	M	91113	O	+	-	140	90	27	12	6	9	92.3	44	71	>1	1.2	36	3.2	Fatty liver	CVT	
37	Ramesh	38	M	91310	O	+	-	138	90	23	10	5	8	112	40	71	>1	1.3	46	3.5	NSY	Young stroke	
38	Chandrakumar	49	M	91752	C	+	-	150	100	32	12	8	12	97.5	48	71	>1	1	48	2.5	Fatty liver	AWS	
39	Marimuthu	46	M	91438	ODW	-	+	100	70	34	12	8	14	102.6	140	71	>1	2.4	168	3	Fatty liver	ACP	
40	Ramamurthy	48	M	113236	O	+	-	130	94	23	12	6	5	104.1	329	109	>2	23.8	109	4.6	Fatty liver	AH	
41	Ravi	52	M	108881	O	+	-	120	70	14	8	6	-	95	38	40	>1	0.9	52	3.2	NS	CAD/ old IWMI	
42	Nuthankumar	37	M	115914	O	-	-	140	100	24	8	6	10	108.7	48	102	<1	1.8	106	4	fatty liver	AG	
43	Mari	37	M	115889	D	-	-	100	70	26	8	8	10	121	44	40	>1	1.9	55	4.3	Fatty liver	AG - DM	

44	**Karthick**	**20**	**M**	**115650**	**S**	**-**	**-**	**100**	**70**	**9**	**5**	**4**	**-**	**96.2**	**52**	**50**	**>1**	**1**	**39**	**3.5**	**NSY**	**Fever**
45	**Sugukumar**	**49**	**M**	**116675**	**O**	**+**	**-**	**190**	**110**	**21**	**7**	**5**	**9**	**106.6**	**66**	**85**	**<1**	**1.2**	**209**	**3.31**	**Fatty liver**	**Acute CVA ICH/SHT**
46	**Dhanasekar**	**40**	**M**	**115895**	**C**	**+**	**-**	**150**	**100**	**32**	**10**	**8**	**14**	**102**	**194**	**86**	**>2**	**1.8**	**166**	**3.4**	**Fatty liver**	**AWS**

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
47	Karthick	32	M	1E+07	D	+	-	130	80	30	10	8	12	93.1	88	66	>1	2.8	56	3	Fatty liver	AWS	NS
48	Venugopal	38	M	115929	D	+	-	130	100	15	7	4	4	87.5	70	52	>1	1.2	52	2	NSY	CVT	NS
49	Feroze	24	M	115203	S	-	-	120	80	12	8	4	-	112.1	45	28	>1	0.8	114	3.9	NSY	poisoning	Self harm
50	Munusamy	47	M	116034	C	+	+	110	70	30	10	8	12	102	346	160	>2	4.9	158	3.6	Fatty liver	AH	Hepato billary
51	Nithyanandan	26	M	106125	H	-	-	100	70	12	8	4	-	112.9	57	35	>1	1.3	49	3.5	NSY	Fever	Infection
52	velu	47	M	106229	ODW	+	-	110	70	24	10	9	5	97.3	86	44	>1	1.2	106	3.2	MLS	Ca Oesophagus	GIT
53	Marimuthu	40	M	108376	ODW	+	-	100	70	32	12	9	11	105.2	114	48	>2	0.8	66	2.5	Fatty liver	AI	NS
54	David	53	M	108470	C	-	-	110	70	24	10	6	8	100.8	55	66	>1	0.8	116	3.8	NSY	AWS	NS
55	Natarajan	50	M	110811	O	+	-	100	70	27	10	9	9	106.5	52	48	>1	2.8	1.7	3.7	DCLD	DCLD	Hepato billary

56	**Padmanabhan**	**47**	**M**	**108492**	**D**	**+**	**+**	**120**	**70**	**20**	**10**	**6**	**4**	**92.6**	**48**	**50**	**<1**	**0.8**	**72**	**2.3**	**NS**	**RVD**	**Infection**
57	**Arumugam**	**42**	**M**	**109652**	**D**	**+**	**-**	**114**	**70**	**23**	**10**	**5**	**8**	**109.7**	**156**	**102**	**>1**	**1.2**	**32**	**3.2**	**CM Liver absess**	**Liver absess**	**Infection**
58	**Ethiyappan**	**26**	**M**	**109772**		**+**	**+**	**100**	**70**	**18**	**8**	**6**	**4**	**88.3**	**42**	**46**	**<1**	**1**	**78**	**3.2**	**NSY**	**RVD**	**Infection**

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
59	Chellappan	48	M	110919	O	+	-	210	130	18	10	4	4	100.2	56	68	<1	0.9	116	3.8	NSY	AHT/Acute CVA	CV
60	Murugan	44	M	103442	D	+	-	120	80	27	7	6	14	91.4	48	44	>1	1	96	4	Fatty liver	Old PTb	Infection
61	Govindasamy	32	M	103537	ODW	-	-	100	60	23	9	5	9	121	52	48	>1	1.2	74	3.8	Ascites	CAD/ECF	CV
62	Ellappan	60	M	103495	O	-	-	150	90	34	12	12	10	102.8	188	36	>2	2.8	196	3	Fatty liver	AI	NS
63	Murugesan	47	M	103868	D	+	-	100	60	33	10	9	14	109	126	42	>2	8.8	226	3.5	DCLD	DCLD	Hepato billary
64	Mani	56	M	103726	ODW	-	-	150	90	16	6	4	6	97.7	56	44	>1	0.7	162	2.8	NSY	CAD/CA	CVS
65	Damotharan	48	M	103786	ODW	-	-	110	70	23	9	8	6	107.8	114	88	>1	1.4	104	2.8	NSY	Growth Oesophagus	GIT
66	Selvakumar	75	M	104276	O	-	-	96	70	16	8	4	4	101	166	82	>1	2.2	112	3.3	NSY	CAP	Infection

67	**Rajendran**	**47**	**M**	**105768**	**D**	**+**	**+**	**120**	**70**	**26**	**12**	**8**	**6**	**86.7**	**58**	**49**	**>1**	**1.4**	**102**	**3**	**Fatty liver**	**CVT**	**NS**
68	**Hamaiyan**	**65**	**M**	**105794**	**O**	**+**	**-**	**100**	**70**	**19**	**10**	**5**	**4**	**102.9**	**172**	**100**	**>1**	**2.4**	**206**	**3.2**	**Secondary Liver**	**Ca stomach Stage IV**	**GIT**
69	**Prabhadoss**	**25**	**M**	**105869**	**S**	**-**	**-**	**96**	**60**	**36**	**12**	**10**	**14**	**92.3**	**486**	**208**	**>1**	**16.7**	**140**	**2.8**	**AL**	**PHF**	**Hepato billary**

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
70	Nagaraj	61	M	108787	O	+	-	114	70	12	6	6	-	97.5	46	26	>1	1	40	3.2	NSY	PTB	infection
71	Kanniappan	55	M		O	+	-	110	70	19	8	5	6	100.2	40	42	<1	1.3	36	2.9	NSY	Pt.Sequale	Infection
72	Pandiyan	40	M	108966	ODW	-	-	130	90	20	10	5	5	97.6	48	40	>1	1.2	78	4	NSY	CVT	NS
73	Somasustham	45	M	111215	O	+	-	120	80	33	12	8	13	101.5	29	26	>1	0.8	78	3.8	Fatty liver	PN	NS
74	Arumugam	40	M	108904	D	+	-	120	70	22	10	6	6	95.3	156	100	>1	2.2	80	3.2	Lt.side PE	Lt.Para pnemonic effusion	Infection
75	Govindan	48	M	111369	O	+	+	150	100	20	10	5	5	108.7	64	48	>1	2.3	40	3	Fatty liver	Seizure	NS
76	Rajasekar	50	M	111195	O	+	-	110	60	32	12	8	12	92.6	402	164	>2	11	179	3.4	Fatty liver	AH	Hepato billary
77	Alakesan	63	M	11377	O	-	-	120	80	24	8	6	10	107.5	62	44	>2	1.8	86	2.5	NSY	PTb	Infection

78	**Selvaraj**	**43**	**M**	**116209**	**C**	**+**	**-**	**140**	**90**	**35**	**12**	**10**	**13**	**107.5**	**41**	**49**	**<1**	**4.8**	**100**	**3.7**	**Fatty liver**	**Werinkey's encephalopathy**	**NS**
79	**Settu**	**56**	**M**	**116214**	**O**	**-**	**-**	**150**	**100**	**30**	**12**	**8**	**10**	**86.9**	**172**	**69**	**>2**	**1.2**	**61**	**3.6**	**Fatty liver**	**AWS**	**NS**
80	**Shankar**	**45**	**M**	**116499**	**O**	**-**	**-**	**130**	**80**	**26**	**12**	**6**	**8**	**112.8**	**58**	**40**	**>1**	**2.2**	**66**	**3.3**	**Fatty liver**	**AG**	**GIT**
81	**Murugan**	**34**	**M**	**86599**	**D**	**+**	**-**	**100**	**70**	**32**	**12**	**8**	**12**	**91.6**	**150**	**94**	**>1**	**0.8**	**119**	**3.5**	**NSY**	**AI**	**NS**

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
82	Kumaravel	26	M	116113	D	+	-	130	80	28	12	8	10	104.5	42	40	>1	1.4	86	3	Fatty liver	CAH	NS
83	Dasaradhan	31	M	118758	O	+	-	120	70	12	4	4	4	97.6	37	39	<1	1	213	3.8	NSY	OPC	Self harm
84	Srinivasan	27	M	110839	O	-	-	100	70	25	6	9	10	87.5	42	50	<1	1.2	106	3.6	NSY	Emphyema	Infection
85	Kesavan	44	M	113828	C	+	-	160	90	22	7	11	4	104.8	68	32	>2	1.6	86	3.1	Fatty liver Gr.I	Young stroke	NS
86	Balachandran	34	M	113550	D	+	-	100	70	17	6	4	7	106.5	48	71	>1	3.1	126	3.2	DCLD	DCLD	Hepato billary
87	Ashok Kumar	35	M	113912	H	-	-	100	60	30	10	8	12	110.1	102	73	>3	16.9	96	3	Fatty liver Gr.II	AH	Hepato billary
88	Stalin	26	M	115785	C	-	-	160	90	32	10	10	12	115	51	72	>2	1.3	96	3	Fatty liver	CS	NS
89	Nehru	57	M	115904	D	+	-	140	90	14	6	4	4	106.5	38	71	>1	1.4	102	3.5	NSY	Bilateral thin SDH	NS
90	Beliappan	60	M	115884	C	-	+	120	80	15	6	4	5	83.7	54	36	>1	2.2	55	3.8	Fatty liver	CAD	CVS

91	**Mohamed Asan**	**49**	**M**	**1E+06**	**C**	**-**	**-**	**130**	**70**	**24**	**8**	**6**	**8**	**109**	**54**	**42**	**>1**	**1.3**	**102**	**3.5**	**NSY**	**AG**	**GIT**
92	**Balamurugan**	**40**	**M**	**116575**	**O**	**-**	**-**	**150**	**90**	**33**	**10**	**9**	**14**	**103.1**	**126**	**103**	**>1**	**1.2**	**103**	**3.6**	**fatty liver**	**PN**	**NS**
93	**Gopi**	**35**	**M**	**116492**	**O**	**-**	**-**	**130**	**80**	**27**	**12**	**8**	**7**	**102**	**112**	**66**	**>1**	**1.6**	**49**	**3.2**	**fatty liver**	**AG**	**GIT**
94	**Venkattaraman**	**46**	**M**		**C**	**+**	**+**	**200**	**120**	**17**	**12**	**5**	**-**	**84.4**	**38**	**36**	**>1**	**1.4**	**52**	**2**	**↑B/L RE**	**AHT-DM**	**CVS**

Sl.No.	Name	Age	Sex	IP/OP No.	Occupation	Personal History		Blood Pressure mmofhg		Total Score (AUDIT)	Audit Score			Investigations								Diagnosis	System wise Diagnosis
						Smoking	High risk sexual behaviour	SBP	DBP		Consumption Score	Alcohol dependence Score	Alcohol detected problem score	MCVin fl	AST in u/l	ALTin u/l	AST/ALT	bilirubin mg/dL	Sr. ALP in u/l	Albumin g/dL	USG abdomen		
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
95	Venu	45	M	1E+06	O	-	-	120	80	24	10	8	6	105.3	40	70	<1	3.4	52	3	Fatty liver	AL	Hepato billary
96	Manikanadan	27	M	109216	O	-	-	120	80	28	12	6	10	105.4	42	46	<1	1.3	28	3.5	NSY	attempted hanging	Self harm
97	Narayanasamy	40	M	109177	O	-	-	110	70	32	10	8	14	108.1	124	69	>1	3.2	46	3.2	Fatty liver	AWS	NS
98	Sakthivel	43	M	76707	C	+	-	140	100	30	12	6	12	102	58	50	>1	1.2	74	3	Fatty liver	AG	GIT
99	Sriram	40	M	78788	D	+	-	130	80	29	10	8	11	106	102	108	>1	1.2	116	3.8	Fatty liver	AWS	NS
100	Karunakaran	52	M	795422	O	-	-	100	60	25	10	7	8	103.2	55	62	<1	2	93	2.9	Fatty liver	Pyopneumo thorax	Infection

Abbreviations:

NSY	Normal Study	CAD	Coronary Artery Disease	PN	Peripheral Neuropathy	U	Umemployed
RE	Renal Echo	NS	Nerves System	ALD	Alcoholic Liver Disease	ODW	Other Daily wagers
AWS	Alcohol Withdrawal Syndrome	GIT	Gastero Interstinal System	AH	Alcoholic Hepatitis	O	Others
AG	Alcoholic Gastritis	DCLD	Decompensated Liver Disease	SVT	Supraventricular Tachycardia		

AI	**Alcoholic Intoxication**	**CVT**	**Cerebral Venous Thrombosis**	**D**	**Driver**
AHT	**Accelerated Hypertension**	**CS**	**Cervical Spondylosis**	**C**	**Construction Workers**
DM	**Diabetes Mellitus**	**OPC**	**Organophosphorus Compound**	**S**	**Students**
PTb	**Pulmonary Tuberculosis**	**RVD**	**Retroviral Disease**	**H**	**Hospitality**

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

EC RegNo.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. M.Raja,
II nd year, MD General medicine
Madras Medical College, Chennai-3.

Dear Dr.M.Raja

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Mean Corpuscular volume as a marker of alcohol use dis-order" No.13072013.

The following members of Ethics Committee were present in the meeting held on 02.07.2013 conducted at Madras Medical College, Chennai -3

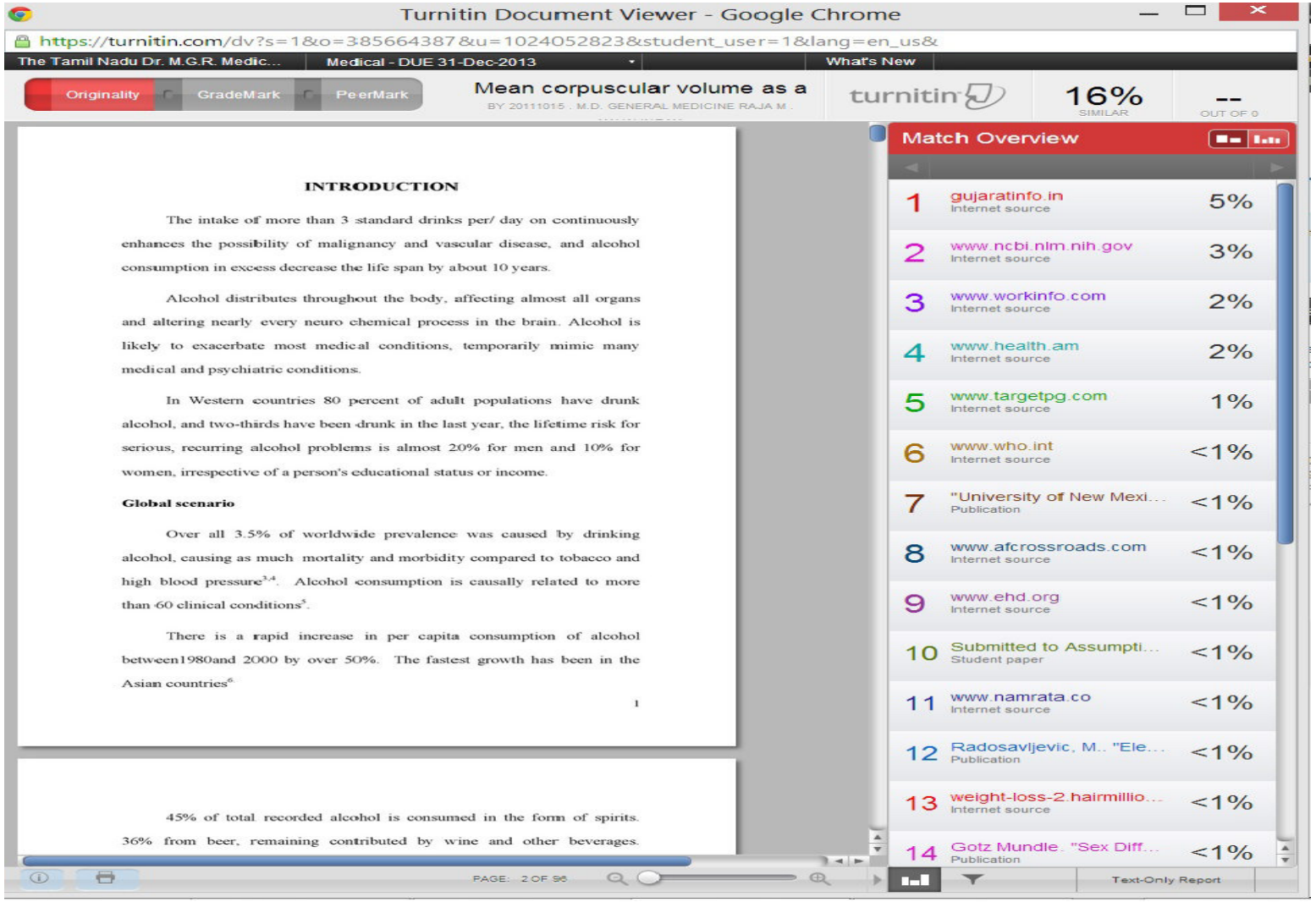
1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL - Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini 12/7/13
Member Secretary, Ethics Committee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INTRODUCTION
The intake of more than 3 standard drinks per/ day on continuously enhances the possibility of malignancy and vascular disease, and alcohol consumption in excess decrease the life span by about 10 years.
Alcohol distributes throughout the body, affecting almost all organs and altering nearly every neuro chemical process in the brain. Alcohol is likely to exacerbate most medical conditions, temporarily mimic many medical and psychiatric conditions.
In Western countries 80 percent of adult populations have drunk alcohol, and two-thirds have been drunk in the last year, the lifetime risk for serious, recurring alcohol problems is almost 20% for men and 10% for women, irrespective of a person's educational status or income.
Global scenario
Over all 3.5% of worldwide prevalence was caused by drinking alcohol, causing as much mortality and morbidity compared to tobacco and high blood pressure[3,4]. Alcohol consumption is causally related to more than 60 clinical conditions[5].
There is a rapid increase in per capita consumption of alcohol between1980and 2000 by over 50%. The fastest growth has been in the Asian countries[6]
1
45% of total recorded alcohol is consumed in the form of spirits. 36% from beer, remaining contributed by wine and other beverages.
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INTRODUCTION The intake of more than 3 standard drinks per/ day on continuously enhances the possibility of malignancy and vascular disease, and alcohol consumption in excess decrease the life span by about 10 years. Alcohol distributes throughout the body, affecting almost all organs and altering nearly every neuro chemical process in the brain. Alcohol is likely to exacerbate most medical conditions, temporarily mimic many medical and psychiatric conditions. In Western countries 80 percent of adult populations have drunk alcohol, and two-thirds have been drunk in the last year, the lifetime risk for serious, recurring alcohol problems is almost 20% for men and 10% for women, irrespective...